UNITED STATES ARMED FORCES
MEDICAL JOURNAL

Volume VII June 1956 Number 6

MANIFESTATIONS AND TREATMENT OF NERVE GAS POISONING IN MAN

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THE effects of nerve gas poisoning described below are those that have been observed in man after exposure to Sarin (brand of isopropyl methyl phosphonofluoridate).[1-4] Exposure to other organic phosphate anticholinesterase compounds, such as parathion, mipafox, tetraethyl pyrophosphate (TEPP), and hexaethyl tetraphosphate (HETP), which have been widely used as insecticides, results in the same manifestations,[5-11] and it is believed that the effects of other nerve gases, such as Tabun (brand of ethyl phosphorodimethylamide cyanidate) would be similar. The manifestations due to these compounds are entirely attributable to the inhibition of cholinesterase enzymes in the tissues, resulting in the accumulation of acetylcholine.[12] The treatment of poisoning caused by any of these compounds is the same, and the measures recommended for the management of nerve gas poisoning are also recommended for the management of poisoning due to other organic phosphate anticholinesterase compounds.

ABSORPTION

The nerve gases may be absorbed through any body surface. Vapor, spray, or aerosol is readily absorbed through the respiratory tract or eyes, resulting in local effects on these tissues. If absorption through the respiratory tract is sufficiently great, this is followed by generalized systemic effects. Liquid nerve gases or solutions may be absorbed through the skin, conjunctivas, or gastro-intestinal tract, producing local and then generalized systemic effects.

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MECHANISM OF ACTION

The effects of the nerve gases are due entirely to their ability to inhibit cholinesterase enzymes throughout the body. Because the normal function of these enzymes is to hydrolyze acetylcholine wherever this compound is liberated, such inhibition results in the accumulation of excessive concentrations of acetylcholine at its various sites of action. These include the endings of the parasympathetic nerves to the smooth muscle of the iris, ciliary body, bronchial tree, gastro-intestinal tract, bladder, and blood vessels; parasympathetic nerves to the secretory glands of the respiratory tract and to cardiac muscle; and sympathetic nerves to the sweat glands. The accumulation of acetylcholine at these sites results in characteristic "muscarine-like" signs and symptoms (table 1). The accumulation of acetylcholine at the endings of motor nerves to voluntary muscle and in the autonomic ganglia results in "nicotine-like" signs and symptoms. Finally, the accumulation of excessive concentrations of acetylcholine in the brain and spinal cord results in characteristic central nervous system symptoms.

The inhibition of cholinesterase enzymes throughout the body by nerve gas rapidly becomes more or less irreversible, so that the effects of these compounds are prolonged. Until the tissue cholinesterase enzymes are restored to normal activity, probably by their regeneration over a period of days or weeks, there is a period of increased susceptibility to the effects of another exposure to any of the nerve gases. During this period the effects of repeated daily exposures by any route are cumulative.

EXTENT OF DEPRESSION OF CHOLINESTERASE

Local ocular and respiratory manifestations may occur without any general depression of the cholinesterase activity in the plasma and red blood cells. If systemic manifestations occur, plasma and red blood cell cholinesterase will be found to be depressed, below 50 per cent of initial level of activity following inhalation of nerve gas vapor, below 30 per cent following ingestion of liquid, and below 15 per cent following percutaneous exposure to liquid. After marked depression, plasma cholinesterase gradually returns to the initial level of activity over a period of 30 days, and red blood cell cholinesterase over a period of 90 days. Following repeated exposure there is no correlation between the onset of symptoms and the precise level of cholinesterase activity of the plasma or red blood cells, which may be markedly depressed without symptoms necessarily ensuing.

EFFECTS OF NERVE GAS VAPOR

Within a few minutes after exposure to nerve gas vapor there appear local effects on the smooth muscle of the eye and respira-

TABLE 1. *Signs and symptoms of nerve gas poisoning*

Site of action	Signs and Symptoms
	Following local exposure
1. Muscarine-like	
Pupils	Miosis, marked, usually maximal (pinpoint), sometimes unequal
Ciliary body	Frontal headache, eye pain on focusing, slight dimness of vision, occasional nausea and vomiting
Conjunctivas	Hyperemia
Nasal mucous membranes	Rhinorrhea, hyperemia
Bronchial tree	Tightness in chest, sometimes with prolonged wheezing expiration suggestive of bronchoconstriction or increased secretion, cough
	Following systemic absorption
Bronchial tree	Tightness in chest, with prolonged wheezing expiration suggestive of bronchoconstriction or increased secretion, dyspnea, slight pain in chest, increased bronchial secretion, cough, pulmonary edema, cyanosis
Gastro-intestinal	Anorexia, nausea, vomiting, abdominal cramps, epigastric and substernal tightness (? cardiospasm) with "heartburn" and eructation, diarrhea, tenesmus, involuntary defecation
Sweat glands	Increased sweating
Salivary glands	Increased salivation
Lacrimal glands	Increased lacrimation
Heart	Slight bradycardia
Pupils	Slight miosis, later more marked
Ciliary body	Blurring of vision
Bladder	Frequency, involuntary micturition
2. Nicotine-like	
Striated muscle	Easy fatigue, mild weakness, muscular twitching, fasciculations, cramps, generalized weakness, including muscles of respiration, with dyspnea and cyanosis
Sympathetic ganglia	Pallor, occasional elevation of blood pressure
3. Central nervous system	Giddiness, tension, anxiety, jitteriness, restlessness, emotional lability, excessive dreaming, insomnia, nightmares, headache, tremor, withdrawal and depression, bursts of slow waves of elevated voltage in EEG, especially on overventilation, drowsiness, difficulty concentrating, slowness of recall, confusion, slurred speech, ataxia, generalized weakness, coma, with absence of reflexes, Cheyne-Stokes respiration, convulsions, depression of respiratory and circulatory centers, with dyspnea, cyanosis, and fall in blood pressure

tory tract, and on the secretory glands of the latter. If the concentration of vapor is sufficiently great, the nerve gas is rapidly absorbed by the respiratory tract and carried throughout the body by the blood, resulting in widespread systemic effects.

Local Ocular and Respiratory Effects. These begin within one to several minutes after exposure, and include pupillary constriction, which may be maximal, conjunctival hyperemia, aching in

and behind the eyes, especially on focusing, slight dimness of vision, frontal headache, watery nasal discharge, and a sensation of tightness in the chest. Occasionally, twitching of the eyelids may occur. These local effects may last from 1 to 14 days.

Systemic Effects. These begin within several minutes. If exposure is lethal, death may occur within several minutes to several hours; if not, symptoms may persist from one to five days.

Muscarine-like effects. The sensation of tightness in the chest increases. Bronchial secretion becomes excessive, and may cause coughing, airway obstruction, and respiratory distress. Salivation increases and laryngeal spasm may occur. If the upper airway becomes obstructed by secretions or by laryngeal spasm, or if the bronchial tree becomes obstructed by secretions or bronchoconstriction, little ventilation may occur in spite of respiratory movements, and cyanosis and unconsciousness may ensue. Following inhalation of nerve gas vapor the respiratory manifestations predominate, but, if the exposure is not so overwhelming as to cause death within a few minutes, other muscarine-like effects appear, including sweating, anorexia, nausea, substernal "tightness" attributable to cardiospasm, abdominal cramps, vomiting, diarrhea, and involuntary defecation and urination.

Nicotine-like effects. With the appearance of moderate muscarine-like systemic effects, generalized weakness and muscular twitching, pallor of the skin, and moderate elevation of the blood pressure occur. Weakness of the muscles of the tongue and pharynx may aggravate upper airway obstruction. If the exposure is severe, there may be generalized paralysis, including the muscles of respiration.

Central nervous system effects. The earliest effects include tension, anxiety, restlessness, insomnia, excessive dreaming, and giddiness. If exposure is marked, headache, tremor, drowsiness, impairment of memory, slowing of reactions, apathy, depression, confusion, ataxia, slurring of speech, coma, disappearance of reflexes, Cheyne-Stokes respiration, convulsions, and central depression of respiration follow. The latter adds to respiratory embarrassment caused by weakness and airway obstruction. The blood pressure falls terminally, probably because of depression of the circulatory center.

EFFECTS OF NERVE GAS LIQUID

Local Effects. The local ocular effects are similar to the effects of vapor. There is no immediate local pain or irritation. Following ingestion of solutions containing nerve gas, the initial symptoms are usually gastro-intestinal. Nerve gas has no taste. Following cutaneous exposure, there is no local irritant change, but local sweating and muscular twitching may occur.

Systemic Effects. These are similar to the effects of vapor, but the sequence and time of onset of symptoms vary with the route of exposure. Respiratory and ocular symptoms are the first to appear after inhalation of vapor, gastro-intestinal symptoms after ingestion of liquid, and sweating after cutaneous exposure to liquid. The onset and progression of symptoms are most rapid after inhalation of vapor and slowest after cutaneous exposure to liquid. The duration of symptoms tends to be longest after the latter. Marked miosis occurs only if there has been local exposure to liquid or vapor.

CAUSE OF DEATH

Death is caused by respiratory failure resulting from weakness of the muscles of respiration, central depression of respiration, and airway obstruction by bronchial and salivary secretions and perhaps by bronchoconstriction. If respiration is maintained artificially and secretions removed by postural drainage and suction, and diminished by atropine, the patient will usually survive unless the exposure has been so overwhelming as to produce depression of the circulatory center, with peripheral vascular collapse and fall in blood pressure.

PREVENTION OF POISONING AND HANDLING OF CASUALTIES

The gas mask must be put on at once on signal, or if any of the following are noticed under conditions of potential exposure: a faint, sweetish, fruity odor; a feeling of tightness in the chest or difficulty in breathing; miosis, eye pain, or unexplained dimness of vision; muscular twitching; or other manifestations of nerve gas poisoning. If possible, the breath should be held until the mask is on and properly adjusted. It then must be worn until chemical test procedures indicate the absence of nerve gas and the all clear signal is given.

The standard (U. S. Army, M9A1) gas mask protects against the effects of vapor, which is not absorbed through the skin to a significant degree in ordinary field concentrations.[13] Liquid nerve gas penetrates rapidly through ordinary clothing or impregnated permeable clothing. However, absorption through the skin requires a period of several minutes, so that, unless the exposure is overwhelming, there should be sufficient time to remove contaminated clothing and to blot and wash away liquid on the skin.

Casualties who have been contaminated with liquid nerve gas may endanger unprotected personnel, so that attendants should wear a gas mask, and, if possible, protective heavy rubber gloves and aprons.[14] If conditions permit, aid stations should be established up-wind from the most heavily contaminated areas. Casualties must be undressed in the open, away from uncontaminated

patients, and contaminated clothing and equipment left outdoors, down-wind from the aid station.

TREATMENT

Life-Saving Measures

Termination of exposure. If the atmosphere is still contaminated a gas mask should be put on the casualty. Clothing contaminated with liquid should be promptly removed, and any liquid on the skin should be blotted with a cloth. The contaminated area should be rubbed with a fresh cloth soaked with water, and then washed with soap and copious amounts of water, if possible. If heavy contamination of a limb has occurred, a tourniquet may be applied just proximal to the contaminated area for 30 minutes, being loosened for half a minute at 10-minute intervals. If liquid nerve gas should get into the eye, instant action is necessary to save life. The head should be tilted back and water poured slowly onto the eye, while the eyelids are held open with the fingers. The patient should be instructed to hold his breath during this procedure, and his gas mask then put on.

Atropine administration. The appearance of any local or systemic signs or symptoms of nerve gas poisoning calls for the immediate injection of atropine. Because this drug inhibits the action of acetylcholine at its many sites of action, other than the voluntary muscles, it has a moderate inhibitory influence on the muscarine-like and central neural effects of nerve gas, but no appreciable influence on weakness and fasciculations. The action of 2 mg of atropine sulfate or tartrate begins 1, 8, or 20 minutes after intravenous, intramuscular, or oral administration, and is maximal in 6, 35, or 50 minutes. The effects of the drug administered in the same dose by any of these routes are about the same after absorption has occurred.

If symptoms of nerve gas poisoning are mild, 2 mg of atropine should be injected intramuscularly and repeated at 20-minute intervals until the muscarine-like symptoms due to nerve gas are relieved and signs of mild atropinization (dry mouth and skin) occur. If symptoms are moderately severe, 2 to 4 mg should be injected intravenously, or, if this is not feasible, intramuscularly, and repeated in doses of 2 mg at 3- to 8-minute intervals until the muscarine-like symptoms are relieved and signs of atropinization develop. If respiratory distress or convulsions are present, the initial dose should be 4 to 6 mg. In severe poisoning the effect of each injection of atropine may last only from 10 to 30 minutes. Atropine administration should be repeated at appropriate intervals to relieve muscarine-like and central neural symptoms due to nerve gas, and to maintain a mild degree of atropinization for at least 24 to 48 hours. In severe poisoning as much as

24 to 48 mg of atropine may be required. Once symptoms have become mild, atropine may be administered by mouth.

In the presence of systemic evidence of nerve gas poisoning there is increased tolerance for atropine, so that fairly large doses may be administered before signs of atropinization appear. However, in subjects who have absorbed little or no nerve gas, 2 mg of atropine produce mild symptoms of atropinization. A second dose of atropine produces moderate, but not incapacitating symptoms, while a third dose, administered within an hour, interferes with ordinary activity. Signs of overatropinization include dry mouth, thirst, hoarseness, dry flushed skin, dilated pupils, blurred vision, tachycardia, urinary retention, constipation, slowing of mental and physical activity, headache, giddiness, disorientation, hallucinations, drowsiness, and sometimes maniacal behavior. Body temperature may increase, particularly in a warm environment or after exercise. While overatropinization may be incapacitating, fortunately only extremely high doses endanger life. To avoid the administration of incapacitating doses of atropine to persons who have absorbed little or no nerve gas, the self-administration of atropine should be limited to 2 mg, and administration by personnel other than a medical officer limited to a total of 6 mg.

Removal of bronchial and salivary secretions. The casualty is placed in the prone position with his head to one side, and the foot of the litter or bed elevated to promote drainage. If airway obstruction occurs the tongue is pulled forward and secretions cleared from the mouth and pharynx manually and, if possible, by aspiration. An oropharyngeal or nasopharyngeal airway may be inserted, but will generally be tolerated only by unconscious or partly conscious patients. If the upper airway remains obstructed in spite of these measures and cyanosis ensues, an endotracheal tube may be inserted and suction carried out through a catheter passed through the tube.

Artificial respiration. If respiration is severely impaired, death will occur in a matter of minutes unless effective artificial respiration is begun immediately and maintained until spontaneous respiration is resumed. In casualties with severe poisoning this may require several hours of continuous artificial respiration. The technics of artificial respiration are described by Elam and associates in an accompanying article.[15] Oxygen should be administered whenever available. While atropine administration facilitates respiratory exchange by suppressing bronchial and salivary secretion and bronchoconstriction, and perhaps by diminishing central depression of respiration, it does not obviate the need for artificial respiration in the patient with severely impaired breathing.

Symptomatic Treatment

Convulsions may not be entirely relieved but can be reduced in severity by adequate doses of atropine. If they should be so prolonged as to interfere with respiration and thereby endanger life, trimethadione (Tridione) should be administered intravenously in doses of 1 gram every 15 minutes up to a maximum of 5 grams. If trimethadione is not available or is ineffective, thiopental sodium may be administered, but care must be taken to avoid overdose and depression of the respiratory center.

Apprehension may be diminished by 0.1 gram of pentobarbital sodium orally, and ocular symptoms may be relieved by the local instillation of 2 per cent homatropine hydrobromide or 1 per cent atropine. Penicillin should be administered to prevent or treat pneumonia in persons who have had severe respiratory depression or increased bronchial secretion.

SUMMARY

The manifestations and treatment of poisoning due to nerve gas are very similar to those of other organic phosphate anticholinesterase compounds, including the insecticides parathion, TEPP, and HETP. The manifestations are entirely attributable to inhibition of cholinesterase enzymes in the tissues, resulting in the accumulation of acetylcholine. Ocular and respiratory effects may follow purely local exposure. Systemic absorption results in accentuation of the respiratory effects, in other muscarine-like symptoms, and in nicotine-like and central neural manifestations. Measures for prevention of poisoning include prompt masking and removal of liquid contamination from skin and clothing. Treatment consists of the administration of large doses of atropine, artificial respiration and oxygen when needed, and alleviation of airway obstruction.

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THE PESTICIDE PROBLEM

"The introduction of pesticides to the agricultural market in ever increasing chemical complexity, quantity, and numbers has created a widespread interest in the possible dangers of consuming foods treated with these chemicals. By and large they are poisons, and injury can be expected when the dose ingested is excessive. Our major pharmacologic problem, however, is not the determination of the rather crude end point represented by death, but rather an evaluation of the continued use of the agricultural products treated with normal amounts of the pesticide. This hazard is dependent more specifically on the injury produced by ingestion of small amounts over long periods of time. We cannot compress into a few months an experiment dealing with a lifetime process. Our greatest problem therefore arises from the fact that evidences of safety must be obtained from the feeding of laboratory animals over their life span and then observing the effects produced by the chemical."

—O. GARTH FITZHUGH
in *Antibiotics and Chemotherapy*
p. 88, Feb. 1956

TISSUE BANK PIONEER HONORED

The Navy League's Annual Award of Merit was presented to Commander George W. Hyatt, MC, USN, by Secretary of the Navy Charles S. Thomas at the Navy League Convention in May. Commander Hyatt was chosen for the award because of the large part he had played in organizing and bringing to its present eminence the Tissue Bank at the National Naval Medical Center, Bethesda, Md.

This project pioneered in developing the freeze-dry process of preserving human tissues so that they could be stored at room temperatures for later clinical use. Short-term preservation in nutrient media at ordinary household refrigerator temperatures is also utilized. A third method of preservation is slow or quick freezing, with or without water binders, for storage at dry ice temperatures until needed.

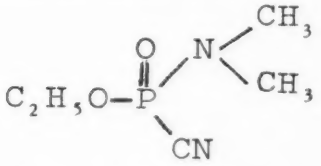
The Tissue Bank has rendered service of the greatest value, especially to military medical facilities but also to other governmental and civilian hospitals in the United States and even abroad. The arteries, bone, skin, dura mater, and other human tissues it has preserved and distributed have made possible many advanced surgical procedures. Large grafts of major arteries have been spectacular, while about 80 per cent of the services of the Bank have been in supplying to orthopedic surgeons material for notably successful bone grafts.

CHEMISTRY, DETECTION, AND DECONTAMINATION OF NERVE GASES

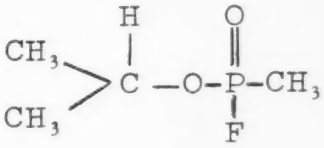
ALBERT A. KONDRITZER

AMONG the most toxic of potential chemical warfare agents are a group of organic esters of phosphoric acid derivatives. They were discovered in Germany prior to World War II through industrial research aimed at finding new and more potent insecticides. The high toxicity of these compounds, which have the property of irreversibly inhibiting the enzyme cholinesterase, brought them under military scrutiny as effective chemical warfare agents. Today a number of related but less toxic compounds, for example, tetraethyl pyrophosphate (TEPP), parathion, and malathion; are widely used in agriculture as insecticides and have caused a number of fatalities as a result of accidental poisoning.

The organic phosphorus compounds that are considered to have possible use as chemical warfare agents have been given the popular designation of "nerve gases," although they are not gases but volatile liquids. The chemical structures of two of these chemicals are given below.



Tabun (brand of ethyl phosphorodimethylamide cyanidate)



Sarin (brand of isopropyl methyl phosphonofluoridate)

CHEMICAL AND PHYSICAL PROPERTIES

Tabun is a colorless to brownish liquid that gives off a colorless vapor having a faint, sweetish, fruity odor. The freezing point is -56°F to -58°F; on heating, decomposition occurs below the estimated boiling point of 459°F. The vapor pressure is 0.070 mm Hg at 77°F. At this temperature a cubic meter of air is saturated by 570 to 610 mg.

From Chemical Corps Medical Laboratories, Army Chemical Center, Md.

Sarin, when pure, is a colorless liquid that is volatile at ordinary temperatures, giving off a colorless and odorless vapor. The freezing point is -36°F and the boiling point 297°F. The vapor pressure is 1.57 mm Hg at 68°F, at which temperature 12 grams saturate a cubic meter of air. Sarin is water soluble, being miscible with water in all proportions.

Tabun and Sarin are unstable in the presence of water. The instability is due to hydrolytic reactions, Tabun forming cyanide as one of its products, and Sarin yielding nontoxic products. The hydrolysis, comparatively slow by water, is catalyzed effectively by hydroxyl ion and much less so by hydrogen ion. In alkaline solutions of pH 12 or higher, hydrolysis is extremely rapid, but between pH 4 and 7 it takes place at such a slow rate as to be of little practical value in destroying either Tabun or Sarin. Other factors influencing the rate of hydrolysis are temperature, quantity of dissolved solids, and concentration of the agent.

DETECTION

Sight and smell are of little value in detecting the presence of the nerve gases. Not only are their vapors colorless; Sarin also is odorless, and Tabun has only a faint fruity odor that may easily be masked by field conditions. In addition, the nerve gases are nonvesicant and nonirritating to the skin, so give no early warning by skin injury. As a result, *early detection by the soldier under field combat conditions is only possible through a knowledge of the signs and symptoms of poisoning,* as described by Grob[1] in the preceding article.

Objective detection of the presence of nerve gas depends on chemical or physical tests. Both Sarin and Tabun can be detected by the reactions discovered by Schoenemann, whereby cyano- or fluoro-phosphonate in the presence of peroxide in alkaline solution oxidizes ortho-tolidine to a dye. The accepted mechanism for this reaction postulates that the nerve gas reacts with perhydroxyl ion to form a perphosphonate intermediate which oxidizes the amine to a colored product. Many compounds capable of yielding colored derivatives under moderate conditions of oxidation have been evaluated at this Center as indicators in the Schoenemann reaction. Of these, indole and ortho-dianisidine have been found suitable for field detection of nerve gases. Physical methods and technics, such as those based on spectrophotometric absorption characteristics, may also be adapted for this purpose.

To differentiate between the two nerve gases, other procedures must be used. Methods have been developed for separately identifying Sarin and Tabun by the reactions of their hydrolysis products. Detection of fluoride ion or hydrogen fluoride identifies Sarin, while Tabun may be identified through the cyanide ion,

hydrogen cyanides, or dimethyl amine. Chemicals, apparatus, and equipment have been assembled into special kits for detecting and identifying the nerve gases, as well as other likely chemical warfare agents, and for collecting samples of these agents for laboratory study.

The Chemical Agent Detector Kit, M9A2, has been designed for field use by combat troops to inform them when the protective mask may safely be taken off or when decontamination of an area has been accomplished after a gas attack. This kit is not intended to warn troops to mask in the event of a gas attack. It is a small, compact, portable unit, operable by one man. It provides for detection within several minutes of dangerous concentrations of nerve gas vapors, by a modification of the Schoenemann reaction that uses indole and sodium pyrophosphate peroxide as reagents. In the event of a positive test for nerve gas, identification of the agent as either Tabun or Sarin can be made with the Chemical Agent Analyzer Kit, M10A1. This is a portable unit developed for use by chemically trained men who are part of a laboratory team. With this kit, the presence of nerve gas in dangerous concentrations is confirmed by means of the color produced with sodium perborate and ortho-dianisidine, a more sensitive modification of the Schoenemann reaction than the one used in the Detector Kit. A cyanide reaction, specifically, color development in gamma-picoline with 1-phenyl, 2-methyl pyrazolone, is used to determine the presence of a cyanide-containing agent, *i. e.*, Tabun. The combination of a positive Schoenemann reaction and a negative cyanide test indicates the presence of Sarin.

DECONTAMINATION

General Principles. Making a contaminated object, person, or area safe for unprotected personnel is accomplished by removal, destruction, detoxication, or covering the chemical agent. The object is to reduce the contamination to a permissible level with the least expenditure of labor and materials and in the time allowed. Decontamination may have to be performed by the individual on himself or his equipment, using whatever is at hand, or it may be done by personnel of his unit, under the supervision of persons specially trained and with material carried by the unit. When possible, however, it is done at decontamination points in maintenance areas, where thorough decontamination can be carried out by well-trained units.

Personal. Liquid nerve gases readily penetrate the bare skin and mucous membranes, and when systemically absorbed can be quickly incapacitating or lethal. In the presence of abrasions or open wounds, which are particularly vulnerable to rapid penetration, even a small dose of agent may be fatal. Decontamination procedures must be applied at once and these must not irritate

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or damage the skin, either physically or chemically. Blotting unabsorbed agent from the skin is desirable, if absorbent material such as a handkerchief or one of the cloths from the Protective Ointment Kit M5A1 is available. Vigorous rubbing of the contaminated area must be avoided, because this will increase the absorption of the toxic agent. In any event, the contaminated area should be thoroughly flushed with a canteen of water. Washing with soap is desirable, especially when the skin is greasy or grimy. Solutions of sodium bicarbonate may be used instead of water for flushing the contaminated skin, but strong alkali and slurries or solutions of strongly alkaline bleach should be used cautiously or not at all. They may injure or irritate the skin and thus increase penetration. For similar reasons, organic solvents should not be used on skin contaminated with nerve gas.

Following decontamination, the area should be examined occasionally for local sweating and muscular twitching. If these occur, 2 mg of atropine should be injected intramuscularly via an uncontaminated area of the thigh or upper arm. The individual should continue his combat or other duties, as systemic symptoms of nerve gas poisoning may not occur or may be mild, if the decontamination was successfully carried out.

Liquid nerve gas in the eye constitutes a serious threat to life; as little as 0.01 ml-drop of Sarin can kill a man of average weight. Owing both to the rapidity and high degree of absorption by the ocular route, steps to remove the agent from the eye must be taken within 30 seconds. The canteen should be opened as quickly as possible, the head tilted back so that the eyes look straight upward and, while the contaminated eye is held open with the fingers, water poured slowly into the eye for not less than 30 seconds. This irrigation must be done in spite of the presence of nerve gas vapor, the breath being held as long as possible. Immediately after decontamination the protective field mask should be put on. During the next few minutes the pupil of the contaminated eye should be watched, and if it gets smaller, 2 mg of atropine should be injected intramuscularly at once. If the pupil does not get smaller, the ocular contamination may not have been by nerve gas and no atropine is needed.

Clothing and Equipment. Personnel who have to handle contaminated casualties and clothing and may thus come in contact with liquid nerve gas should wear protective clothing and protective masks. The impermeable protective apron (Apron Toxicological Agents, Protective) is intended for such use. The special purpose permeable protective outfit and impermeable protective gloves should be worn when the apron is used. Protective items that may be used in conjunction with the apron include protective mask, hood, and either rubber boots or leather boots treated with protective ointment. Rubber boots, however, should be worn when

contact with liquid nerve gas is anticipated. In an emergency when impermeable protective clothing is not readily available, the protective apron and glove may be worn over regulation clothing, but such an outfit must be worn with caution because it will not give a level of protection comparable to the full protection outfit.

At medical installations, prevention of injury to patients and medical attendants from clothing, blankets, or other equipment that has been contaminated with nerve gases is essential. Contaminated clothing and equipment should be removed from the casualty at the earliest practical moment. Casualties in clothing or blankets known to be contaminated should not be admitted to or removed from medical installations or other inclosed spaces, as this may result in serious injury to other patients and medical personnel, who may come in contact either with the liquid agent or the vapors given off by the contaminated materiel.

During land, air, and sea combat operations, decontamination of aprons and gloves will ordinarily not be possible either at aid stations or by medical units. Impermeable protective equipment which has become contaminated should be placed in a gas-resistant sack or gastight container for later decontamination.

Heavily contaminated impermeable aprons may be decontaminated by immersion in water at a temperature just below boiling for a period of one hour, after which they are dried in air. If the contamination is light or caused by vapor, the apron may be aired in the sun or wind for several days. The same principle applies to impermeable gloves; if they are heavily contaminated, immerse in boiling water for two hours, dry, and return to service. Care must be taken that the gloves are filled with water and that they are kept below the surface of the boiling water.

Gas masks that have been grossly contaminated with persistent nerve gases should be discarded. Masks that have been exposed to droplets or vapor may be decontaminated in an emergency by soaking the rubber parts of the mask in boiling water for four to eight hours. If the contamination has been with vapor only, the gas mask may be soaked for two hours at room temperature in water that has been made alkaline by the addition of sodium carbonate. After rinsing in water, the masks are dried and returned to service. The metal canister may be decontaminated by washing with soap and water or an alkaline solution of sodium carbonate. Gas masks carriers, first-aid pouches, and other web and canvas equipment may be decontaminated by soaking in boiling water for one hour. Shoes, straps, and other leather equipment may be decontaminated by soaking in water heated to a temperature of 122° to 131°F for four to six hours.

The following Department of the Army Technical Manuals were freely drawn upon in the preparation of this report, and may be consulted for a more detailed discussion of the principles and procedures described: TM 3-215, Military Chemistry and Chemical Agents, August 1952; TM 3-220, Decontamination, October 1953; TM 3-290, Individual Protective and Detection Equipment, September 1953; and TM 8-285 (NavMed P-5041, AFM 160-12) Treatment of Chemical Warfare Casualties, 1956.

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SCIENTIFIC LINGO

"We should demand that technologic words never be used in scientific papers if familiar words can be used instead. To give an example, I showed my colleague, the Professor of Anatomy, the following passage: 'hypokalaemic hypochloraemic alkalosis was present.' He had no idea what it meant. Had the statement read: 'plasma potassium and chloride were diminished, CO_2 increased,' he would have understood. Here the simpler phrase is seven letters more. But editors are not always so particular in curbing verbosity. Moreover, another example from a recent paper by a very able and highly cultured author, 'bilateral nephrectomy was performed,' is not only less easy for a non-biologic scientist to understand than 'both kidneys were removed,' but is also 11 letters longer . . . . When we use a technologic term there is a strong tendency for the idea to become isolated and inaccessible to those periodic spring cleanings to which we subject our more ordinary mental furniture. Thus, by using a technologic term we often assume an entity where in fact none exists; the next process is to invent it. Thus, by using a technologic term, we may create an artefact."

— GEORGE W. PICKERING, M. D.
in *Annals of Internal Medicine*
pp. 926-927, Nov. 1955

ARTIFICIAL RESPIRATION FOR THE NERVE GAS CASUALTY

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SUCCESS in resuscitation of nerve gas casualties will depend on advance planning, assembly of special equipment, and training of personnel in selected methods of artificial respiration, as well as on recognition of the physiologic responses to anticholinesterase compounds and the modifying effects of atropine. The purpose of this report is to recommend procedures for artificial respiration and to discuss their rationale.

Although there is a paucity of information regarding severe nerve gas poisoning in man, there is considerable experience with human exposures to anticholinesterase insecticides.[1] In general, the effects of severe exposure to these related agents are strikingly similar to the responses of animals to nerve gases, except for differences in the rate of absorption and progression of symptoms.[2,3] To a large extent, therefore, our concepts of therapy have been derived not from experience with nerve gas poisoning in man but from what is considered to be parallel information.[4]

Grob[5] has reviewed the mechanisms, symptomatology, and antidotal therapy of anticholinesterase agents in an accompanying article. From the standpoint of interference with normal respiration, we must consider a wide spectrum of cholinergic effects ranging from a mild sensation of tightness in the chest to an acute fulminant course characterized by unconsciousness, convulsions, bronchoconstriction, pulmonary vascular congestion, nasal and tracheobronchial hypersecretion, profuse salivation, emesis, laryngospasm, and apnea. The tempo of development of these severe manifestations depends on the amount and route of exposure, and on the rate of absorption of nerve gas vapor or liquid. Atropine in sufficient dosage will minimize some of these complications and will control others. In animal experiments, however, atropine, except in very large doses, has failed to bring about prompt reversal of central nervous system effects

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and the early return of spontaneous breathing. In any event, the proper management of artificial respiration requires anticipation and preparation for coping with all of the ventilatory hazards.

In considering the treatment of nerve gas casualties, two limiting facts must be emphasized: (1) The number of severe exposures may preclude effective care for all casualties, and (2) the severe and the moderately severe exposures will necessitate skilled and efficient attention by trained personnel. It seems that a minimum of one attendant per severe casualty will be required to effect successful resuscitation unless tracheal intubation is employed. This estimate is based on realistic experience with paralyzed, comatose, apneic patients in a general hospital. Maintenance of a clear upper airway, a prerequisite for any means of artificial respiration, demands virtually constant surveillance.

The management of the airway and of artificial respiration for field and hospital situations will be considered separately, because of great differences in availability both of special equipment and of specialized personnel.

PRINCIPLES OF EMERGENCY FIELD THERAPY

There are five essential principles that must be applied as expeditiously as possible: (1) protection, (2) decontamination, (3) atropinization, (4) assurance of patency of the airway, and (5) lung ventilation. The exposure should be regarded as severe if the casualty exhibits respiratory difficulty, convulsions, or loss of consciousness.

It has been estimated that with massive vapor exposure to Sarin (brand of isopropyl methyl phosphonofluoridate) the following symptoms will appear within 5 to 15 minutes:

Within 30 seconds	Tightness in the chest
	Dizziness
	In-co-ordination
Within 1 minute	Loss of consciousness
	Prostration
	Pinpoint pupils
Within 5 minutes	Copious salivation
	Generalized muscular twitching
	Convulsions
	Obstructed upper airway
	Hypertension
	Flaccid paralysis
	Respiratory failure
Within 5 to 15 minutes	Cyanosis
	Circulatory collapse
	Death

Procedure. Don gas mask. If feasible, transfer casualty to a noncontaminated area. For decontamination of liquid Sarin see the preceding article by Kondritzer.[6] Atropine should be administered immediately if available.[5] *Clear the pharynx of secretions quickly and inflate the lungs.* Continue artificial respiration until spontaneous breathing returns, unless circulatory failure has been established by cessation for five minutes of the carotid pulse and audible heart beat. Artificial respiration will have to be interrupted repeatedly for quick removal of additional mucus in the upper airway, until salivation has been checked by the action of atropine. The administration of atropine, preferably by intravenous route, should be repeated as long as salivation persists.[5]

With the onset of atropinization, the task of artificial ventilation becomes much easier. Less force is required to inflate the lungs, the airway remains dry, and the casualty resembles an anesthetized paralyzed patient in the operating room. He will probably require artificial respiration for a period of from 30 minutes to several hours, despite sustained atropinization. When spontaneous breathing returns he must be observed vigilantly, because he may lapse again into apnea. Should acute gastric dilatation develop incident to artificial respiration, the stomach must be immediately decompressed.

FIELD MANAGEMENT OF THE AIRWAY

Patency. Apart from the choice of methods to be employed for artificial respiration, the patency of the casualty's airway must be established and maintained. This primary requisite to successful resuscitation cannot be overemphasized.

The jaw muscles are apt to be in tonic or clonic spasm during the early phase following massive vapor exposure to Sarin. The casualty may resemble the patient in a grand mal seizure with the base of the tongue and epiglottis occluding the upper airway. In addition the pharynx becomes filled with thick salivary secretions. The high metabolic rate associated with convulsions accelerates the development of a deep cyanosis.

As soon as the operator is able to manipulate the upper airway, the oral cavity should be cleared of mucus. This is most rapidly accomplished by holding the mouth open and wiping the throat with cloth-covered fingers. The use of a catheter and syringe is theoretically sound, but probably too dilatory in practice. After quick removal of the major pool of secretions, the lungs should be inflated. Further cleaning of the airway is then carried out without inordinate periods of apnea.

Artificial Airway. The spastic tone of the jaw and pharyngeal muscles usually gives way to flaccid paralysis. At this time one of several artificial airways may be used. These include

the oropharyngeal airway, the nasopharyngeal airway, and the tracheotome. None of these artificial airways guarantee a patent airway in the casualty. Insertion of an airway should not be attempted if the airway musculature is in spasm.

The oropharyngeal airway, if employed, must be of the proper size for the casualty. Its position must be checked, care being taken to ensure that the base of the tongue is elevated without the epiglottis being displaced posteriorly or against the larynx. This artificial airway should be changed for a smaller size or eliminated entirely if its presence evokes laryngospasm, stridor, or partial obstruction.

Frequently a nasopharyngeal airway relieves the obstruction without further complicating the problem, provided its tip lies in the pharynx and not in the esophagus. Recently improved nasopharyngeal airways have rounded instead of beveled tips and are less traumatic. These newer nasopharyngeal airways are less apt to produce bleeding from the nasal mucosa, a disadvantage of the older units.

In severe exposures the critical interval during which ventilation of the lungs is the essence of recovery precludes the use of conventional tracheotomy. The less severe casualties who may be observed for the gradual development of airway obstruction will probably be manageable by repeated removal of secretions from the air passage. In such persons the prone position with head down promotes gravity drainage of accumulating secretions.

The rapid tracheotomy technic of Shelden and associates,[7] employing their tracheotome, makes this procedure feasible as an emergency one for the paralyzed cyanotic casualty. In addition, some means is required for inflating or diffusing air or oxygen into the lungs. Shelden and associates stated that the tracheotome can be safely inserted in less than 30 seconds and that it could easily be used in the armed services by hospital corpsmen.

Support of the Mandible. To ensure airway patency and competent fit of the mask, the operator must elevate the mandible of the paralyzed casualty. This manual task necessitates continual attendance. In persons with flaccid muscles the temporomandibular articulation becomes freely mobile and allows the mandible and attached soft parts of the pharynx to move posteriorly and occlude the pharynx (fig. 1A). Repositioning of the mandible by manual traction is easily accomplished by applying forward traction to the angles of the mandible. Bilateral traction applied at points just below the ear lobes frequently results in temporary subluxation of the temporomandibular joint (fig. 1B). This purposeful subluxation, which affords optimal clearance of the ante-

rior soft parts of the pharynx, is not associated with damage to the joint or its ligaments. The most advantageous and efficient position of the operator's hands for support of the airway and mask is indicated in figure 1C. Force is required to secure the fit of the mask and effect a seal under the higher inflating pressures necessary to transfer air to the casualty's lungs. Equally

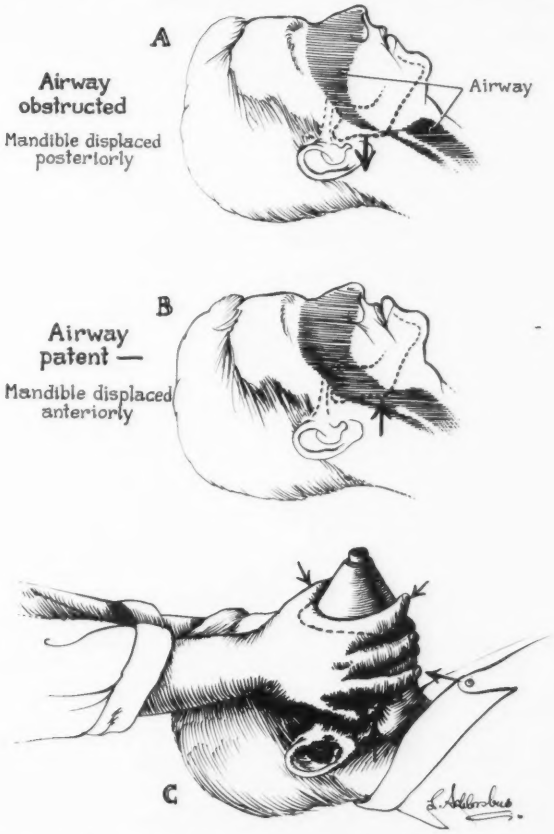

Figure 1. Support of airway and mask. The upper airway of the paralyzed person becomes obstructed primarily due to loss of tone in muscles which normally maintain the position of the mandible. (A) *With relaxation the mandible slides posteriorly allowing the anterior soft parts (tongue, epiglottis, and pharyngeal muscles) to approximate the posterior pharynx.* (B) *Bilateral manual traction at the angles of the mandible (see arrow) elevates these structures.* (C) *This position must be maintained with the application of a mask. Strapping the mask on the head and neglecting this manual traction allows the airway to occlude as in* (A).

important is the opposing force to elevate the mandible, in order to prevent obstruction. This requirement, in particular, renders tight strapping of a mask by means of a harness around the head likely to produce occlusion of the airway if the mandible is not manually elevated. Proper position and support of the jaw usually suffices to maintain patency of the airway without the use of an oropharyngeal airway.

FIELD METHODS OF ARTIFICIAL RESPIRATION

Manual Methods. Although recent revision of the manual methods of artificial respiration[8-15] has improved their effectiveness, there is some doubt that any of the recommended procedures will provide sufficient force to ventilate the apneic nerve gas casualty. Depending on the severity of bronchoconstriction, thick secretions, and vascular engorgement of the lungs, it is anticipated that man may develop severe respiratory hindrance. Experiments in laboratory animals suggest that pressures as high as 50 millimeters of mercury may be required to effect transfer of air to the alveoli. The management of casualties resulting from severe exposure to Sarin vapor will require methods of artificial respiration capable, if necessary, of delivering the higher pressures efficiently. Should it be possible, however, to achieve good ventilation of the lungs by any manual method, this choice provides the simplest scheme for working in a contaminated area, because both operator and casualty may wear their standard gas masks.[16] On the other hand, should experience indicate the manual methods to be ineffective, two alternative procedures are available.

Hand-Operated Bellows. In skilled hands, this device is capable of developing the required pressures. It may be adapted for safe use in a contaminated area by adding a canister at the intake valve and making provision to prevent the recovering casualty from inhaling through the expiratory passage. The latter is not protected by a check valve in conventional models. The hand bellows is preferred by many anesthetists for emergency use and undoubtedly performs well in their hands, but the less experienced operator may expect considerable difficulty in holding the rigidly-coupled bellows-mask assembly on a mobile, mucus-laden, bearded face with one hand. We have observed that when such an operator attempts to deliver a volume by compressing the bellows with his other hand, either the mask rocks off the the face to leak excessively or the force applied to compress the bellows is transmitted to the subject's mandible, producing occlusion of the airway. However, it is well established that with sufficient practice the hand bellows may be very effectively used to achieve good artificial respiration. To overcome the disadvantages of this unit for the neophyte, redesign of the mask-bellows coupling is required.

Mouth-to-Mask and Mask-to-Mask Methods. These modifications of the ancient procedure appear to be especially promising for management of the nerve gas casualty (figs. 2-4). The operator has both hands available to hold the mask tightly and to elevate the airway (fig. 1C). He is more intimately aware of the status of his efforts than when he executes either a manual method (which removes him considerably from direct observation of

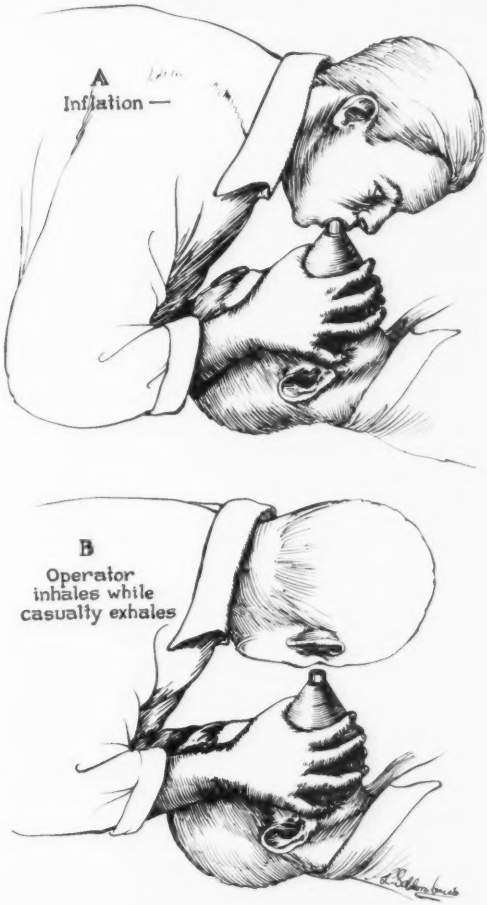

Figure 2. Emergency mouth-to-mask resuscitation (clean atmosphere). (A) *The operator inflates the lungs of the casualty until visible expansion of casualty's chest occurs.* (B) *While the casualty exhales, the operator takes an inspiration of ambient air of about twice his normal tidal volume.*

the chest) or the hand bellows. He can feel whether or not air moves into the chest. With each inflation of the casualty's lungs he can sense the pressure required and the presence of airway obstruction resulting from secretions or malposition of the tongue or epiglottis. Appropriate equipment for performing this method in a contaminated area is currently being developed. Until it becomes available, such an item may be improvised from a stand-

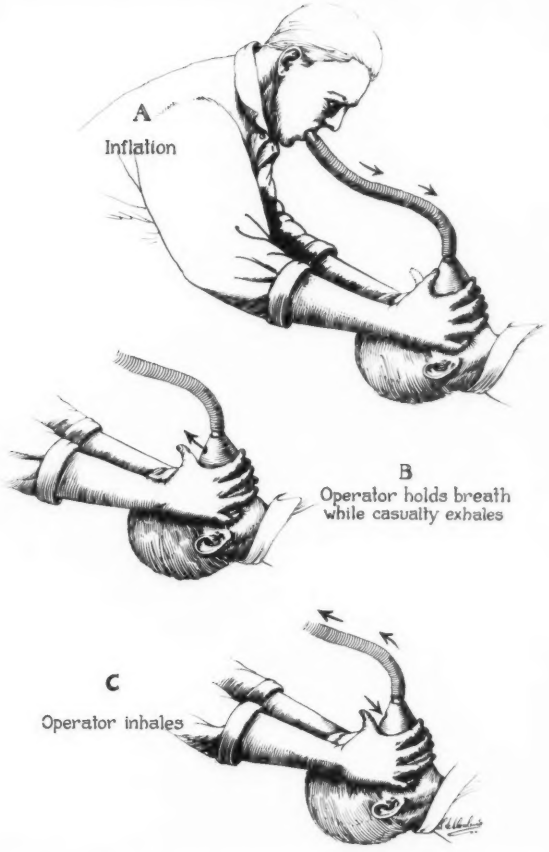

Figure 3. Mouth-to-mask resuscitation for prolonged periods. This modification ensures freedom from severe hypocapnia and associated dizziness of the operator, making it possible to continue artificial ventilation for several hours. The operator holds his breath between inflations while he allows the casualty to exhale through the opening in the mask. After the casualty's exhalation is completed, the operator inspires through the tubing.

ard gas mask, a "mouthpiece" insert made of rubber tubing, a segment of breathing tube, and a rigid oronasal mask of the type used in anesthesia or on resuscitators (fig. 4C). The breathing

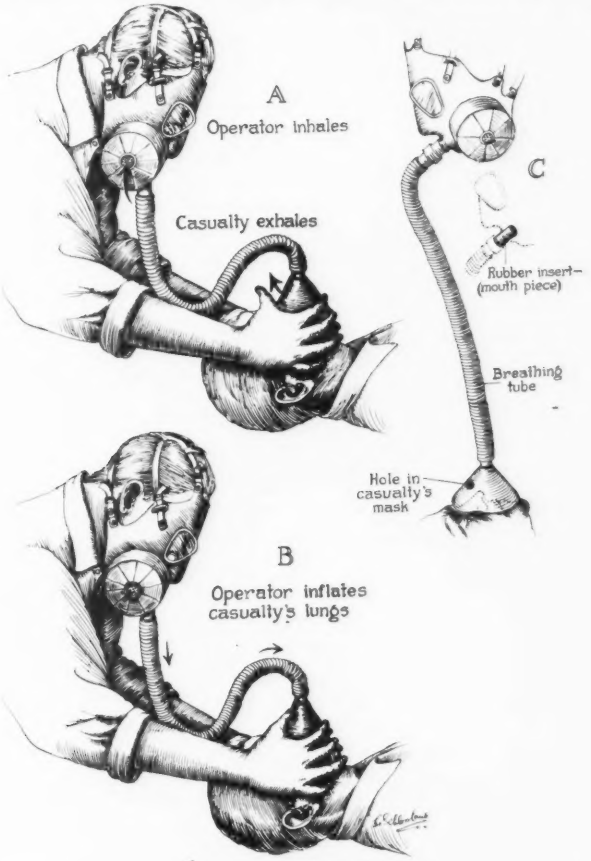

Figure 4. Improvised equipment for use of mouth-to-mask method in a contaminated atmosphere. An extension tubing of about 300-ml volume is attached to the expiratory outlet of the M-9 protective gas mask and to an oronasal mask. A half-inch hole is placed in the oronasal mask in a position convenient for covering with the operator's thumb. This improvised assembly is completed by inserting a relatively stiff segment of rubber tubing three-fourths inch in diameter into the internal part of the expiratory port to serve as a mouthpiece (C). *The cycle for this modification permits the operator to inhale while the casualty exhales* (A) *and to inflate the casualty's lung with the thumb occluding the hole in the mask* (B).

tube, about 1 inch in diameter and 2 feet long, connects the expiratory outlet of the gas mask to the oronasal mask that is intended for the casualty. In the latter mask, a hole about one-half inch in diameter is cut to serve as a valve, which the operator occludes during inflation by means of his thumb and opens to allow the casualty to exhale passively. This sequence is illustrated in figure 4. Because standard gas masks show excessive outward leak at intramask pressures above 20 cm of water, it is necessary to slip a short segment of three-quarter inch, stiff, rubber tubing inside the expiratory outlet of the mask to serve as a mouthpiece around which the operator can effect a satisfactory seal (fig. 4C). When the casualty has been evacuated to a clean area, the mouth-to-mask method may be simplified by elimination of the breathing tube and operator's gas mask (fig. 2). Should the period required for artificial respiration be prolonged, the operator may experience dizziness associated with the excessive blowoff of carbon dioxide. Provision may be made to prevent this effect by using the breathing tube in the pattern shown in figure 3.

The efficacy of the mouth-to-mouth modifications has been found to be remarkably good, provided one important detail is observed: the operator must increase his breathing, or hyperventilate, while performing the method.[17] It is only in this way that he is able to compensate for the lower oxygen and higher carbon dioxide concentrations in his expired air. If he approximately doubles his normal ventilation, satisfactory respiratory exchange in the casualty is obtained.

Tests performed on models have demonstrated that untrained operators can carry out the required 15 to 20 inflations per minute for periods up to an hour, without undue fatigue. The human chest is surprisingly well set up for doing pressure-volume work. The models used for these studies simulated the maximal increases in pulmonary resistance and the maximal decreases in pulmonary compliance anticipated in severe nerve gas poisoning in man.[18] Operators of the mouth-to-mask method performed on this model complained, not of fatigue of the chest, but of the hands. The greatest difficulty encountered was that of holding the mask snugly on the face of the test manikin. This practical problem is common to all the methods considered feasible for artificial respiration in the field, in which higher pressures will be required. It is apparent, in attempts to master this limitation, that the operator can markedly increase his efficiency if he can have both hands to hold the mask and support the airway. For this reason, the mouth-to-mask methods would seem to have preference over other methods.

One of the most difficult and disconcerting problems is apt to be acute gastric dilatation. The presence of bronchoconstric-

tion, necessitating delivery of gas at higher inflating pressures, favors the passage of air through the less resistive path down the esophagus. Animals poisoned by nerve gas, in which the mouth-to-mask methods were used, demonstrated rapid inflation of the stomach during the initial efforts to "open up" the constricted bronchi. Subsequently it was found possible by less zealous "blowing" to ventilate the lungs without inflating the stomach. Pressure applied over the cricoid cartilage may help to prevent gastric dilatation. If pressure is applied over the epigastrium, the operator must be alert to detect silent regurgitation of stomach contents, which may easily be forced into the tracheobronchial tree.

PRINCIPLES OF THERAPY IN A HOSPITAL

Ideally a treatment facility should be set up that provides for the emergency use of a variety of measures. Equipment for endotracheal intubation, mechanical resuscitators, efficient aspiration pumps, and wide-bore catheters (at least one quarter inch in diameter) should be immediately available and operable. For the less severely exposed, the body respirator may be useful. In the very mild cases, oxygen therapy and atropinization alone may suffice.

Atropinization may be more effectively established by intravenous administration. Tonic or clonic convulsions with rigidity of the thoracic musculature constitutes a serious impediment to ventilation and causes increased oxygen demand. Attempts to control such effects with atropine are not apt to be successful and may lead to overatropinization. Grob[5] recommends the intravenous use of trimethadione as an anticonvulsant in preference to thiopental sodium because of its lesser respiratory depressant effect. The latter may be used, however, if trimethadione is not available, but should be given in small doses. The intravenous use of thiopental sodium in doses less than 25 mg has been observed to produce prompt control of chest muscle spasm in dogs poisoned with Sarin.

MANAGEMENT OF THE AIRWAY IN A HOSPITAL

Patency. Secretions may be cleared efficiently by means of a suction catheter introduced through the nose or mouth. Because the major secretions are salivary, the resort to endotracheal intubation is particularly sound, especially if artificial respiration also is required. As mentioned earlier, postural drainage may suffice to eliminate secretions until atropinization supervenes.

Tracheal Intubation. This procedure is recommended in preference to tracheotomy because of a lesser likelihood of aspiration of secretions into the tracheobronchial tree. A cuffed endotracheal

tube affords a convenient coupling for several methods of artificial ventilation. Intubation should not be attempted in the presence of an active gag or laryngeal reflex, and under such circumstances is probably unnecessary. Use of the curved McIntosh laryngoscope blade and a tracheal tube with moderate curvature, facilitates intubation. The vocal cords should be visualized; it is unwise for the nonexpert to attempt "blind" intubation. Some atraumatic material, such as a roll of gauze, should be used as a bite block to prevent the recovering casualty from occluding the tracheal tube. The tube should be well anchored by means of adhesive tape at the mouth to prevent its moving either out of the larynx or into a bronchus.

The particular advantage of tracheal intubation is that it would allow, along with the use of an automatic resuscitator, the adequate coverage of a number of apneic casualties by one skilled operator.

METHODS OF ARTIFICIAL RESPIRATION IN THE HOSPITAL

Bag-Mask, Bellows-Mask, and Mouth-to-Mask Procedures. Any of these simple technics is sound for emergency treatment of apnea, provided the operator is dextrous in its use. None of these methods is ideal for long-term artificial respiration in a well-equipped hospital.

The bag and mask, a standard item among anesthetists, consists of an oxygen supply, face mask, and attached rubber breathing bag that is manually compressed. Oxygen or air should be introduced at a point between the patient and the bag at a flow of from 10 to 15 liters per minute. Outflow of excess gas through a "tail" on the bag minimizes build-up of carbon dioxide in the bag. The rate of leak from the "tail" is adjusted with a hemostat or screw clamp. Compression of the bag to produce visible chest expansion ensures adequate ventilation. Expiration occurs passively with release of bag pressure.

The bellows-mask assembly has the particular advantage of portability without an oxygen source.

The mouth-to-mask procedure is as immediately available as a simple face mask alone, which may be carried in one's pocket. For short-term emergency use, the problem of excessively lowering the carbon dioxide blood content of the operator is negligible; hence the breathing tube may be omitted.[17]

Body Respirator. If available, this method has the advantage in the less severely exposed cases of permitting free access to the pharynx for removal of secretions, as compared with devices that involve the use of an oronasal mask. However, for dealing with the anticipated problems of bronchoconstriction

and pulmonary vascular congestion, the body respirator suffers the handicap of inadequate reserve inflating pressure. If additional inspiratory force is required beyond that available with the body respirator, a variety of accessory supplemental methods may be employed. An oxygen-fed bag may be coupled to the mask or tracheal tube and manually compressed in rhythm with the inspiratory phase of the respirator. The same results may be obtained by the mouth-to-mask procedure. Temporary use of a pressure-cycled resuscitator in addition to the body respirator is another alternative. In this connection, an important phenomenon of breathing mechanics deserves emphasis: in the presence of bronchoconstriction or reduced lung compliance, one inflation of the lungs facilitates subsequent inflations. This effect is especially pronounced in Sarin poisoning. Thus, a few high-pressure inflations often "open up" the lungs so that normal inspiratory pressure then produces adequate ventilation for several minutes. Pulmonary tension regenerates gradually and ventilation decreases until a few more inspiratory surges are needed. This sequence recurs until adequate atropinization ameliorates the pulmonary anticholinesterase effects.

Mechanical Resuscitators. Until recently these pressure-cycled devices also were incapable of providing sufficient inflating pressure in the presence of an increased respiratory impedance. Conventionally the suck and blow resuscitators operate between fixed pressure limits (+14 and −9 millimeters of mercury) and cycle rapidly when breathing is obstructed, giving inadequate lung ventilation. A higher pressure range of operation (+27 and −2 millimeters of mercury) has been recommended for potential use in the nerve gas casualty. In addition, a manually controlled adjustment with which the inflating pressure can be set temporarily at +35 millimeters of mercury has been recommended. These higher pressures should be employed only until expansion of the lungs is achieved, in view of their potentially deleterious effects on lungs and circulation and the hazard of gastric dilatation.

SUMMARY

Artificial respiration for the nerve gas casualty presents a major challenge. The complications of copious salivation and obstruction of upper airway by flaccid muscles impose serious limitations. Moreover, bronchoconstriction and "stiffening" of the lungs are anticipated from Sarin studies in animals. These complications render manual methods of artificial respiration difficult or ineffective. Given time, atropine in sufficient dosage will largely eliminate these pulmonary complications; meanwhile, mouth-to-mask or mask-to-mask methods are capable of overcoming them. Whatever method of artificial respiration is em-

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ployed, the complication of acute gastric dilatation is likely unless a conservative schedule of initial inflations is used. In the organized treatment facility, the use of endotracheal intubation and mechanical resuscitators capable of delivering higher pressures are indicated.

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PSYCHIATRIC SCREENING OF COMBAT PILOTS

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AMONG the many variables that must be considered in aircrew effectiveness is personality. The affective life of an individual has much to do with the levels of stress which he can endure, the motivation with which he attacks a goal, and the perseverance of his efforts in the face of hazards. It may be maintained that among the most fundamental determinants of combat superiority are personality factors; but even if this is true, we know far more about other factors in combat effectiveness than we do about emotion. Men who reach the stage of aerial combat see well enough to fight, fly well enough to fight, and have the physical endurance, at least for most of the missions in Korean combat. Selection in the Air Force in this area is rigorous and effective. However, the question every battle commander must ask himself is how emotion will affect the behavior of his men.

A survey of the famed 51st fighter wing (the Mig-killers) revealed, during one phase of the war, that about 50 per cent of their F-86 pilots had never fired their guns. Of the nearly 50 per cent who had fired their weapons, only about 10 per cent had ever hit anything. The basis for the difference between the 10 per cent effectives and the remainder includes, along with superior vision and other variables, personality functions for the combat task of a superior but as yet undetermined sort. What these personality functions or psychodynamics are, precisely, is at present only vaguely understood. If human beings were robots, one could measure their strength, plot their course, and predict accurately the outcome of battle in terms of the physical factors involved. Since they are not, and since we at present know so little about these affective factors, some very surprising things occur in the combat world. So much so, that combat commanders in the past have been known to conclude that only the acid test of combat itself will satisfactorily determine the behavior of their men under combat. Men who have been shy and timid have developed into "tigers," and men who have all their lives bullied and dominated others about them have cowered in fear and panic. Individuals

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who previous to combat have had rather stable personalities have found themselves filled with crippling anxiety in combat; men tormented with fears and tensions have found deep gratifications in the release of aggression. Through combat, individuals who previously had never been bothered with interpersonal conflicts have found themselves violently hostile to leaders and colleagues.

One of the major difficulties facing the investigator of behavior under such conditions is the fact that combat itself is subject to variable types of emotional stresses. Few men can view combat objectively. As in other areas of human behavior where emotions are involved, the investigator must watch lest his own security needs color his findings. An act of heroism is a remarkable event in human affairs. Glory and honor surround it. Yet those who study combat know that sometimes men are heroes as a result of feats performed in states of panic and fear. For this and other reasons, it is most difficult for researchers who have never seen or experienced combat to understand the nature of the stresses involved.

PAST RESEARCH EFFORTS

The Air Force Medical Service has for several years conducted research on the problem of the psychiatric casualties that occur under combat stress in aerial warfare. This research effort has been largely directed toward the obtaining of group psychologic test data which could be used in conjunction with clinical appraisals for the screening of individuals who were likely to do poorly or to break down under the stresses of training and/or combat. Something over 5,000 men have been studied at cadet level, and predictions were made as to how each of these men would perform under training and/or combat stress.

As adequate criterion data were lacking, predictions were made largely from hypotheses which were presumably based on evidence obtained from the psychiatric studies of World War II combat pilots. The hypotheses used are of the sort which reflect traditional military and psychologic concepts of stability and endurance factors in personality. The hypotheses included, as positive for combat success, evidences of maturity, stability, and responsibility—traits that would ordinarily make up what might be thought of as an ideal officer and gentleman. Negative factors were evidences of psychopathology, neurotic tendencies, instability, or lack of responsibility, that is, behavior that would make up what would usually be thought of as typical of disturbed, psychopathic, or immature individuals. Most of the traits used in these predictions were of a surface sort, and deeper dynamics of personality were largely ignored.

FOLLOW-UP STUDY

In February 1953, a research team was organized and sent to the area of the Korean conflict to follow as many of these 5,000

pilots into the combat zone as possible, in order to determine their proficiency under combat stress.* To our knowledge, this may be one of the few occasions where men who had been studied previous to combat were then followed up in combat to determine their success at their tasks.

One hundred and eleven of the previously studied 5,000 pilots were located and studied in the combat zone. The data obtained included combat proficiency ratings by squadron commanders, operations officers, and peers; objective performance data from the squadron files; and clinical appraisals of proficiency in combat, plus any information available on the men's emotional life. Only four of these 111 pilots were found to be actual psychiatric failures in combat. Of the four failures, one was a psychopath, one had a severe anxiety reaction, one was an alcoholic, and one was at least a borderline psychopath who, concluding after the first mission in combat that people "got killed doing this," asked for and obtained release from his combat duties. The investigators attempted to make as thorough a study as possible of each pilot where found in the combat zone. The data thus obtained concerning each pilot's proficiency in combat has since been correlated with the previously obtained test results and the predictions made previously for these same individuals. Sufficient data for analysis was obtained for 65 of these pilots. Interestingly enough, the top jet ace of the study (a double ace) was a predicted failure. The four failures were all predicted successes. A correlation between the predictions of the training level psychologists and the combat proficiency estimates of the combat psychologists was -.43, significant at the 5 per cent level of confidence.

The pilot Stanine test yielded only one coefficient (+.35) of marginal significance (10 per cent level of confidence) between combat psychologists' final appraisals and Stanine scores. Correlations of peer-superior ratings of flying proficiency in training and combat were significant at the 1 per cent level of confidence between training upper classmate ratings and combat peer-superior ratings on the trait of likeability (+.57) and between training classmate ratings of flying proficiency and the combat psychologists' final ratings of proficiency in combat (+.52). Previous

*Maj. Jack Kavanagh, USAF (MC), then Head of the Department of Psychiatry, School of Aviation Medicine, Randolph Air Force Base, Tex., and Maj. John Mebane, USAF (MC), also of the same department, served as consultants to this team, and each spent one month in the combat zone. The team was composed of a clinical psychologist and a senior flight surgeon who was a psychiatrist and formerly wing surgeon of the famed 51st fighter wing at K-13 in Korea. The latter, Dr. Frederick J. Hinman, now psychiatrist at Veterans Administration Hospital, Denver, Colo., contributed much to the success of this mission. These two men, assisted by a Medical Service Corps officer and an enlisted man, traveled extensively throughout the combat zone carrying out these criterion studies during the last six months of the Korean conflict.

psychologic test data, clinical appraisals, and predictions failed to correlate at a significant level of confidence with either the objective combat data or the presumably valid estimates of squadron commanders, operations officers, and peers in combat.

However, the correlation between the combat psychologists' estimates of proficiency and the mean combat proficiency ratings of squadron operations officers, commanders, and flying colleagues was +.78 (1 per cent level of confidence); that between the combat psychologists and the training psychologists was -.32 (5 per cent level of confidence) (table 1).

TABLE 1. *Correlations between proficiency estimates for 65 combat pilots in the Korean conflict*[1]

Combat proficiency rating traits	Peer-superiors vs. combat psychologists		Peer-superiors vs. training psychologists	
	Coefficient of correlation	Significance*	Coefficient of correlation	Significance*
Competence	+.65	1	−.26	
Fairness	+.54	1	−.38	
Courage	+.80	1	−.13	
Responsibility	+.51	1	−.20	
Likeability	+.68	1	−.29	10
Discipline	+.55	1	−.24	
Mean	+.78	1	−.32	5

* Per cent level of confidence.

RESULTS

The results seem to indicate that the psychologists at training level understood neither the nature of combat stress and its effect on personality nor the psychic variables which constitute probable success or failure. The fact that their estimates were in significant disagreement with the estimates of both combat peer-superiors and combat psychologists indicates that the hypotheses on which their predictions were made were significantly in error. It is important to note also that the predictions were made in the opposite direction. The fact that the combat psychologists' estimates agreed in general with the peer-superiors' estimates indicates a most essential need for investigators in this area to have a realistic understanding of combat, since training researchers tended to predict successes to fail and failures to succeed. The results support the hypothesis that predictions of combat proficiency, based on psychiatric data adequately oriented to combat, can be made with sufficient precision to be of value in screening.

The fact that only four of 111 pilots did fail indicates, however, that whatever screening technics were being used for these pilots were functioning rather well.

It is recommended that adequate criterion data for this research effort be obtained. The significant (1 per cent level) positive correlations between the clinical appraisals of the combat psychologists and the peer-superior appraisals indicate that there may be cause for hope that it would be possible, given sufficient insight into personality as involved in combat stress, to do a better job of psychiatric screening for combat duties than has been done heretofore. The combat data obtained in Korea certainly suggest that there is a real question as to whether stability as conceived of in civil society is an adequate basis for psychiatric screening for combat duties. Investigations should be made concerning the specific effects of intense, prolonged fear and of how they involve their unconscious companion, anxiety; as well as of the effects of intense, prolonged hostility and release of aggression, and of how they involve their unconscious companion, guilt. How do these emotions affect the structure and stability of the personality as the ego strives to maintain integrated behavior for survival and success at combat? From this perspective, some men with rather disturbed personalities may be more insulated against crippling anxiety in combat than are some normal persons, even though the combat data from Korea indicates that pilots without symptoms tended to do better.

If it is true, as postulated here, that we do not as yet understand many of the basic psychodynamics of combat proficiency, how then are we to screen out those predisposed to breakdown? The present situation seems to be that we are only able to identify those who prove themselves unable to carry out their duties for psychiatric reasons, or appear to evidence a high immediate probability of not being able to do so. Even here caution is called for. In this study there were a number of highly disturbed individuals who not only were carrying on but were doing so in a superior manner, and who finished their tours successfully, although the cost to them in anxiety was very high. In some instances the very anxiety involved appeared to contribute considerably to the performance of the pilot. In this regard it was interesting to note that some men with obsessive-compulsive personalities did very well in B-29's, but none were seen to do well in fighters; conversely, a number of those with "psychopathic personalities" did splendidly in fighters but were not seen to do well in bombers. Possibly there are elements of stress which vary from task to task, so much so that men with certain personality syndromes are better equipped for certain types of combat flying.

In the Korean conflict, as in all wars fought by the United States, great men appeared on the horizon of combat: Gabreski,

Fernandes, Fisher, Jabara, McConnell, and others too numerous to mention here. Sometimes outnumbered, they met the Mig—sometimes with the F-86, sometimes in a slow, aged bomber, and sometimes in a "chopper." Their record is one in which they defeated the enemy with amazing scores. It was our great privilege to have known them. We could not fail to note that against the enemy's superior numbers in the north, these superior pilots from the south won and kept control of the North Korean sky, all the way to the Yalu.

SUMMARY

Psychiatric screening for combat duties, based on training level group psychologic test data and/or clinical appraisals as used in the project discussed here, appears as yet unable either to screen out the failures or to identify the successes in combat flying. At present, careful, "on-the-spot" psychiatric evaluations by a psychiatrist, collaborating with a flight surgeon who lives and flies with these pilots, can probably give us the best psychiatric opinion available on the combat proficiency of a given pilot on a given combat tour or mission. Beyond this we cannot go very far with certainty.

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WATER FLUORIDATION AT ARMY POSTS

Fluoridation of drinking water at 21 Army posts in the United States, Alaska, Hawaii, and Puerto Rico has been approved as of 2 April 1956, it was announced by Major General Silas B. Hays, Surgeon General, United States Army.

The fluoridation of drinking water at Army installations lacking the natural fluorine necessary for oral health was approved by the Army Medical Service in July 1954. Through the co-operation of the Corps of Engineers, the fluoridation equipment and engineering skill required to install the process are assured. Each military post must obtain both the approval of The Surgeon General of the Army and the endorsement of the Corps of Engineers.

Regulations now in effect to control the granting of requests for fluoridation have a global application. The program is expanding and no change in Army policy is anticipated in the foreseeable future.

EPIDEMIOLOGIC STUDIES OF HISTOPLASMIN SENSITIVITY

HARRIS D. RILEY, Jr., *Captain, USAF (MC)*

THE HISTORY of histoplasmosis may be arbitrarily divided into two periods. The first began in 1906 when Darling,[1] in Panama, described three cases of illness caused by *Histoplasma capsulatum,* which he thought to be a protozoon. In this period the disease was regarded as extremely rare and invariably fatal. The organism was cultivated and identified as a yeastlike fungus by DeMonbreun[2] in 1934. The second period began in 1941 with the development of a specific cutaneous test.[3] This intradermal histoplasmin test opened new channels of investigation which resulted in the discovery that the infection commonly exists as a widespread but subclinical and benign disease.

The enigma of pulmonary calcification in tuberculin-negative individuals had stimulated wide interest and caused doubt to be cast on the assumption that only in the very exceptional case would a person previously infected with tuberculosis fail to react to strong concentrations of old tuberculin.[4-9] The enigma was solved in 1945 when Christie and Peterson[10,11] demonstrated an immunologic relationship between histoplasmin sensitivity and pulmonary calcification that has been confirmed repeatedly.[12-17] From these and other studies, evidence has evolved that infections with *H. capsulatum* may be benign and asymptomatic and rarely recognized clinically. Reports of vital statistics[18,19] also point up the important role of histoplasmosis. In 1946-1948 no deaths due to this infection were recognized, but 33 fatalities were reported in 1949. It was estimated in 1948 that in children in the East Central United States, histoplasmosis (as indicated by a positive skin test) is almost 10 times as prevalent as tuberculosis.[20]

Many studies, such as that of Mochi and Edwards,[21] on the prevalence of histoplasmin sensitivity in all parts of the world have been reported. Most of these investigations, however, have concerned adults and almost exclusively residents of one particular geographic area. Few studies have concerned individuals

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coming from areas of low prevalence of histoplasmosis into one of high prevalence, and no such study specifically concerning infants and children has been reported.

The pediatric dependents of military personnel arriving at this Air Force base in middle Tennessee afforded an opportunity to study the problem, because Tennessee, especially middle Tennessee, is an endemic area for histoplasmosis with a high prevalence rate of histoplasmin sensitivity.[13,14,22,23] The children studied had lived in all parts of the United States and in some cases in other countries, and had then moved into this area of high incidence. In addition to histoplasmin sensitivity, the prevalence of tuberculin sensitivity in this heterogeneous group of children was determined.

PLAN OF STUDY

For approximately a one-year period, children* who reported to the military dependents clinic of this hospital for medical attention were given intradermal tests with histoplasmin and old tuberculin, and a detailed residence history from birth to the time of testing was recorded.

Subjects. A total of 663 subjects between the ages of birth and 15 years were tested on their first visit. No attempt was made to include children from specific age groups; however, infants below 1 year of age were not tested routinely, and only 28 are included in the total. The majority of the subjects were in the age group of 1-7 years, because the fathers were young military personnel and their children tended to be younger than the clientele of the average civilian pediatric clinic.

Location of Study. This study was conducted in the northwest section of Rutherford County, which lies in the so-called Central Basin, a fertile farming area with rolling terrain and several rivers and streams. The average altitude is about 600 feet, the average annual rainfall 49 inches, and the average relative humidity 67 per cent. Figure 1 depicts the geographic relationships of the locale.

Antigen, Method of Testing, and Interpretation of Tests. Histoplasmin in 1:100 dilution, obtained from the filtrate of a culture of *H. capsulatum*,** and old tuberculin 1:1000 dilution were used in 0.1-ml doses throughout the study, employing the technic of testing described by Christie and Peterson.[10,11] Only fresh

*"Children" also includes infants below one year of age, unless specifically stated.

**Obtained from Department of Pediatrics, Vanderbilt University Hospital, Nashville, Tenn. This dilution is equivalent to 1:1000 of histoplasmin (Lilly). For comparison with other histoplasmin antigens, see Shaw, L. W.; Howell, A., Jr.; and Weiss, E. S.: Biological assay of lots of histoplasmin and selection of new working lot. *Pub. Health Rep.* 65: 583-609, May 5, 1950.

batches of histoplasmin and tuberculin of known potency were used. Separate needles and glassware were used for each antigen, and the equipment was cleaned and autoclaved separately to avoid subsequently using a syringe and needle for histoplasmin testing that had been used previously for tuberculin.

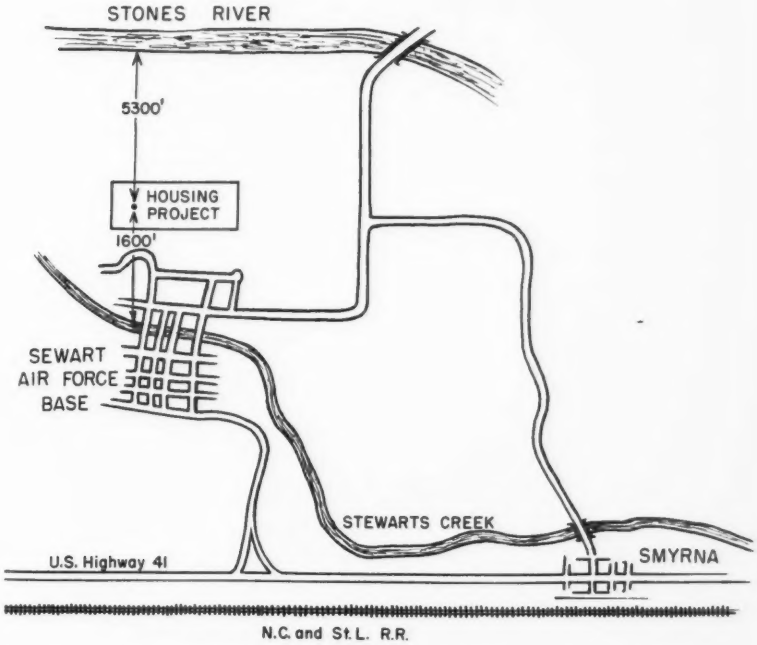


Figure 1. The geographic relationships of the locale in which the study was conducted.

Tests were read in 48 hours, by the author in the majority of the subjects. A positive reaction to either test was considered as one in which erythema and induration measured 5 mm or more. If either test was negative after 48 hours, the parents were instructed to bring the child to the clinic at 72 hours after testing, in case there was any evidence of erythema or induration. If either test was negative at 48 hours but unequivocally positive at 72 hours, it was recorded as positive.

Rigid criteria were used in classifying reactions and all equivocal tests were repeated, usually on the opposite forearm. If this was definitely positive, it was recorded as an initial positive, the original borderline test not being considered as an

"initial test" nor the second test as a "repeat." Those tests that remained equivocal are not included in this report.

In the great majority of instances, the subjects were tested within a short time after arrival in this locale, chiefly because of a local base regulation strongly urging typhoid and tetanus immunizations for all children as soon as possible after arrival.

Most of the test subjects resided in a housing project adjacent to the hospital (fig. 1) and could easily return for interpretation of skin reactions. In the few cases where it was impossible for the child to return at 48 hours, the parents were carefully instructed in drawing on a form the size of the reaction at the end of 48 and 72 hours. Comparison in a pilot group of the parent's interpretation and those of the author indicated satisfactory correlation. If the parents had any doubt as to the reaction, they were asked to have a physician interpret it or to return to the clinic.

Residence History and Regional Classification. At the time of the initial testing, a detailed residence history was recorded giving the exact locations of residence since birth, the length of residence in each site, and the date of arrival in this locale. Adequate residence data was obtained on 583 (87.9 per cent) of the 663 children. At the initiation of the project, the United States was divided into regions (fig. 2), a modification of the geographic divisions used by Palmer[17] and by Beadenkopf et al.[14] If a child had spent as much as 70 per cent of his life in one particular region, he was recorded as "having spent the majority of his life" in that region. Those who had spent less than 70 per cent of their lives in any one region were placed in a special classification and the duration of stay in each region recorded. This group was not included in the category of known residence data but was combined for purposes of analysis with the group of "no record" in tables 1 and 2. Of the 583 subjects on whom adequate residence data was obtained, 498 had spent 70 per cent of their lives in one particular region, and 85 had lived in several regions but not as much as 70 per cent of the time in any particular one. The percentage chosen to represent "majority of life" is a purely arbitrary one, but is approximately the same as that used in other similar types of investigation.[17, 24]

Retesting for Histoplasmosis. Because of the obvious interest in determining the rate of acquisition of the infection by persons who, in many cases, had come from an area of low endemicity of histoplasmosis into a known endemic area, 234 of the subjects who had been negative on initial testing were retested at various intervals. The results of this phase of the investigation will be reported separately.

Roentgenographic and Serologic Studies. A roentgenogram of the chest was obtained on each patient who had a positive intradermal reaction to either tuberculin or histoplasmin. This was repeated as clinically indicated. In a few selected cases, histoplasmin complement fixation studies also were performed.* These determinations could not be made at the military installation where the study was conducted, and due to technical difficulties in transporting specimens too few were obtained to be statistically significant.

RESULTS AND DISCUSSION; HISTOPLASMOSIS

Age Factors and Geographic Influences. The rates for histoplasmin sensitivity reported in table 1 are not as high as those found in children of the same age range *residing* in the central section of the United States,[13-16, 22, 25] because a large number of our subjects came from areas where histoplasmosis is virtually nonexistent and were tested soon after arrival. We tested only 28 infants below the age of 1 year, but found no positive reactors, as compared with an incidence of 1.9 per cent[22] for residents of adjoining Davidson County and of 26.6 per cent[13] for residents of Williamson County, which also adjoins the county in which our study was conducted. Our rates of 1.2 per cent at ages 1-2, 13.4 per cent at ages 2-5, and 18.3 per cent at ages 5-10 contrast with rates for Davidson County *residents* of 18.7, 27, and 50, respectively.[22] In all studies, the sensitivity rates have been observed to increase with age, and are higher in young adults than in children living in the same locale.[15, 17, 25-38]

It is important that the rate of histoplasmin sensitivity be correlated not only with age but also with site of residence. The exact geographic distribution of histoplasmosis has not yet been determined, but fairly well defined endemic foci have been outlined, although the subjects of most previous studies have been adults. In the United States the infection, as determined by skin sensitivity, is highly prevalent in the central portion of the Mississippi River Basin. This region corresponds closely to the geographic distribution of pulmonary calcification.[14, 16, 17, 21, 22, 39-41] Some lesser endemic foci exist in the Middle Atlantic States, in the Potomac and St. Lawrence River Valleys, and in sections of North Carolina. The disease has also been reported from Central and South America and less frequently from Europe, Africa, and the Far East (fig. 3). In parts of Tennessee, Missouri, Kentucky, Illinois, and adjoining states a high percentage of the population are histoplasmin positive by the time they reach puberty, while the further removed from the central Mississippi River Basin the less frequent histoplasmin sensitivity be-

*Through the courtesy of the Laboratories, Department of Pediatrics, Vanderbilt University School of Medicine, Nashville, Tenn.

comes.[14,16,17,21,22,39,42] Distinct intra-state differences have been described.[17,22,26,36,38,77]

TABLE I.
HISTOPLASMIN SENSITIVITY, INITIAL TESTS

Classification	Number Tested	Histoplasmin Positive	
		No.	%
Total	663	93	14.0
Sex			
Male	341	45	13.2
Female	322	48	14.9
Age			
<1 year	28	0	0.0
1 year	83	1	1.2
2 years	232	31	13.4
5 years	262	48	18.3
≥10 years	58	13	22.4
Residence in Region IV *			
<1 year	157	7	4.5
1 year	138	17	12.3
2 years	78	18	23.1
3 years	49	13	26.5
4 years	26	6	23.1
≥5 years	50	11	22.0
No Record	165	21	12.7
Major Region of Residence **			
U.S.A., I	18	1	5.6
II	20	3	15.0
III	50	11	22.0
IV	257	44	17.1
V	91	10	11.0
VI	27	2	7.4
Foreign ***.	35	1	2.9
No Record	165	21	12.7

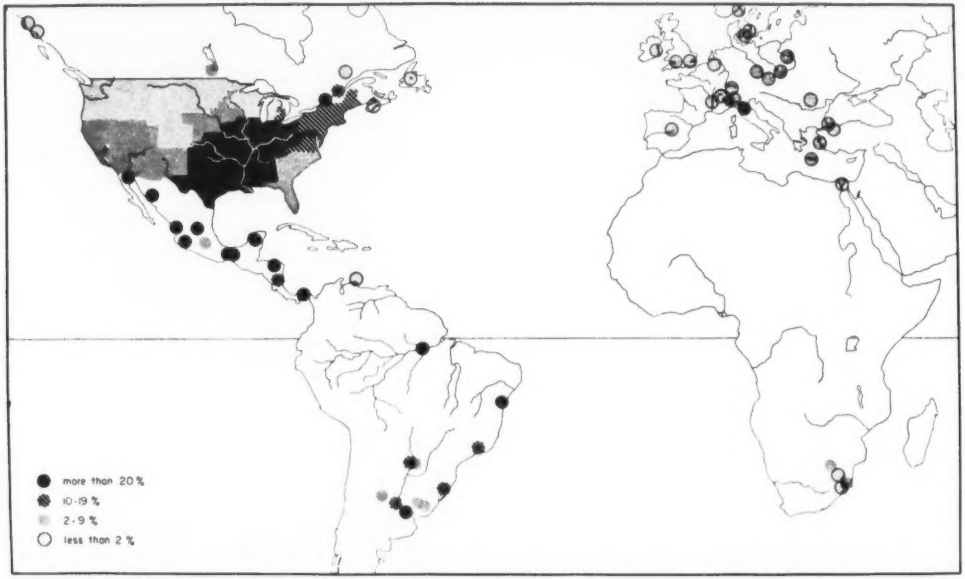
* Region IV is composed of the states of Tennessee, Kentucky, Ohio, Indiana, Illinois, Missouri, and Arkansas. (see figure 2).

** For states contained in the various regions, see figure 2.

***The 35 children with major foreign residence are detailed as follows: 13 Germany, 4 Newfoundland, 3 Alaska, 3 Hawaii, 3 Japan, 3 Canada, 2 England, 1 Brazil, 1 Panama, 1 Okinawa, 1 South Pacific.

From table 1 it is seen that children who had spent the major portion of their lives in states of the western Appalachian slope and states bordering the Mississippi and Ohio Rivers had a higher prevalence of histoplasmin sensitivity than those coming from other areas. This is most notable with respect to previous

Figure 2 Geographic division of the United States which was used in this study.



Each study is indicated by a circle shaded to represent the percentage of positive reactors reported; "less than 2%" includes completely negative findings; areas from which no studies have been reported are unshaded (including the continents of Asia and Australia, which are not shown). Nationwide mapping of sensitivity is indicated for the USA.

—Reproduced from Mochi, A., and Edwards, P. Q.: Geographic distribution of histoplasmosis and histoplasmin sensitivity. *Bull. World Health Organ.* 5: 259-291, 1952.

Figure 3. Geographic distribution of histoplasmin sensitivity throughout the world.

residence in Regions III and IV. Of 50 subjects from Region III, 22 per cent were histoplasmin positive, whereas of 257 from Region IV, the area of highest prevalence in most studies, only 17.1 per cent reacted.* However, breakdown by age groups reveals that the median age of the subjects from Region IV was 4.1 years, whereas the median age for those from Region III was 5.8 years. As there were many more infants in the group from Region IV than Region III (60 as compared to 4), and as the frequency rate of histoplasmin sensitivity is a function of increasing age, the difference becomes more apparent. For children over 2 years of age the rates were 27.3 per cent for Region IV and 21.6 per cent for Region III, and there were no reactors in the combined total of 64 individuals from Regions III and IV below 2 years of age. Furcolow and associates[15, 25] showed that even in areas of high histoplasmin sensitivity, nonlifetime residents may show a higher rate of reaction than lifetime residents.

The number tested from Regions I and II was small, and the 15 per cent of reactors from Region II as compared to only 5.6 per cent from adjacent Region I may be related to age. The median age of the Region II group was greater—6.5 years as compared to 4.3 years for Region I. All three reactors from Region II were over 4½ years of age and all had spent over a year in Tennessee at the time of initial testing, while the only reactor from Region I had not spent any significant period in Region IV prior to testing. The possibility of cross reactions with other fungus diseases, especially coccidioidomycosis, was considered, because all three children from Region II had previously resided in California or Arizona where this disease is endemic,[42] but all three were negative to coccidioidin.

There were 35 children who had spent as much as 70 per cent of their lives in countries outside of the United States before coming to middle Tennessee. The only positive reactor to histoplasmin was a 3½-year-old child who had always lived in Germany before coming to this locality. Histoplasmin skin test 9 days after her arrival and 16 days after her entry into the United States was strongly positive. Twelve other children whose major residence was Germany failed to react.

Table 2 shows the considerable increase in histoplasmin sensitivity rate with increasing length of residence in Region IV, as well as with increasing age. The population studied was

*As determined from five of the more extensive studies,[16, 17, 22, 24, 42] the order of rank of frequency of histoplasmin reactors of the states comprising Region III is: Kansas, 6th; Oklahoma, 10th; Texas, 14th; Iowa, 16th; and Nebraska, 20th. Region IV: Missouri, 1st; Kentucky, 2d; Tennessee, 3d; Indiana, 4th; Arkansas, 5th; Ohio, 8th; and Illinois, 9th.

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TABLE 2

Histoplasmin Sensitivity, Initial Tests, For Age Groups: By Length Of Residence In U.S.A. Region IV By Major Regions Of Residence

	Age, <2 Years			Age, 2 Years			Age, ≥5 Years			Total		
	Number Tested	Positive No.	Positive %	Number Tested	Positive No.	Positive %	Number Tested	Positive No.	Positive %	Number Tested	Positive No.	Positive %
Total	111	1	0.9	232	31	13.4	320	61	19.1	663	93	140
Residence in U.S. Region IV												
<1 year	48	0	0.0	46	3	6.5	63	4	6.3	157	7	4.5
1 year	31	0	0.0	55	7	12.7	52	10	19.3	138	17	12.3
≥2 years	-----	---	---	70	13	18.6	133	35	26.3	203	48	23.6
No Record	32	1	3.1	61	8	13.1	72	12	16.7	165	21	12.7
Major Region of Residence												
U.S.A., I-II	4	0	0.0	15	1	6.7	19	3	15.8	38	4	10.5
III	4	0	0.0	12	2	16.7	34	9	26.5	50	11	22.0
IV	60	0	0.0	97	17	17.5	100	27	27.0	257	44	17.1
V-VI	9	0	0.0	30	2	6.7	79	10	12.7	118	12	10.2
Foreign	2	0	0.0	17	1	5.9	16	0	0.0	35	1	2.9
No Record	32	1	3.1	61	8	13.1	72	12	16.7	165	21	12.7

relatively transient. Only 203 of the 498 for whom residence data was known had spent more than two years in Region IV, but their sensitivity rate was 23.6 per cent as compared with 4.5 per cent for those who had spent less than one year in this endemic area. Length of residence in Region IV did not become a factor, however, until the subject was two or more years of age; none of the 79 subjects less than two years old were positive reactors, even though they had resided in Region IV since birth. This is in contrast to the high rates of sensitivity in native infants reported from adjoining Williamson County.[13]

Table 3 further confirms the effect of the geographic residence of a subject on his likelihood of being sensitive to histoplasmin. Children whose major residence before moving to Region IV had been in Region III or Region V tended to have the highest rates of sensitivity during their first two years of residence in Region IV.

Sex and Race. There was no significant sex variation; 13.2 per cent of the 341 males and 14.9 per cent of the 322 females reacted to histoplasmin. Zeidberg, Dillon, and Gass[13] also found no sex difference, whereas Furcolow and associates,[15, 28] Emmons, Olson, and Eldridge,[43] and Prior, Wilce, and Palchanis[44] reported a predominance of male reactors. None of the 10 Negroes tested in this study were reactors. The number is too small to be significant, but the finding is in line with those of Zeidberg, Dillon, and Gass[13] and Furcolow, High, and Allen[15] who reported a lower percentage of reactors among Negroes.

Comparison With Other Studies. Potency of the antigen, testing technic, and interpretation of skin reactions affect testing results and make comparison of various studies difficult. In this study very rigid criteria for positive reactions were used, and any borderline cases are omitted from the analysis. Another factor of vital importance that was ensured in this study is elimination of contamination of glassware and needles with tuberculin. The importance of those factors in a comparison of sensitivity rates is obvious, but they cannot be readily assessed. It is believed, however, that the difference in reaction rates between other studies of middle Tennessee children[13, 22] and the present one is related primarily to the previous site of residence of our subjects. We found that children who have previously resided in areas where histoplasmosis is uncommon are less likely to exhibit histoplasmin sensitivity when tested soon after arrival in an endemic area than are natives of the area.

Roentgenographic Pulmonary Findings. The relationship of pulmonary calcification to histoplasmin sensitivity has been demonstrated repeatedly, and no attempt was made to analyze

TABLE 3. HISTOPLASMIN SENSITIVITY BY LENGTH OF RESIDENCE IN VARIOUS REGIONS

Major Region of Residence	Length of Residence in Region IV								
	< 1 Year			1 to 2 Years			< 2 Years		
	No. Tested	No. Pos.	% Pos.	No. Tested	No. Pos.	% Pos.	No. Tested	No. Pos.	% Pos.
I	15	1	6.7	3	0	0	0	-	-
II	7	0	0	12	3	25.0	1	0	0
III	25	3	12.0	18	5	27.8	7	3	42.9
V	46	2	4.3	29	3	10.3	16	5	31.2
VI	14	0	0	6	2	33.3	7	0	0

the correlation in this study, although a roentgenogram of the chest was obtained on all positive reactors to either histoplasmin or tuberculin. All types of roentgenographic findings due to histoplasmosis, ranging from mediastinal lymphadenopathy through frank pneumonic infiltrations, both minimal and massive, to the "stippled lung" of miliary calcification, were observed. The findings were in agreement with the results of others that, in this area, pulmonary calcification is much more commonly associated with sensitivity to histoplasmin than with sensitivity to tuberculin.[13, 20, 22, 39, 40, 45, 46] Acquisition of histoplasmin sensitivity can occur at an early age[13, 47] but pulmonary calcification due to histoplasmosis is rare in early life and increases with advancing age. However, Peterson and Christie[40] observed the development of pulmonary calcification in a 4½-month-old infant dying with histoplasmosis. A young child observed in the present investigation exhibited calcification 4½ months after inception of a serologically proven Histoplasma infection.

Cross Reactions. It has been demonstrated that cross-reactions between histoplasmin and other fungus antigens can occur, but need not be a problem.[42, 43, 48, 49] *Candida albicans* is a relatively common cause of oral infection in infants, and if this fungus infection were significant in producing cross reactions a large number of infants would be expected to react to histoplasmin; however, in this study as well as in others,[15, 22] infants have shown an extremely low reaction rate. It has been conclusively shown that no cross reactivity between histoplasmin and tuberculin exists.[50]

In this study, two histoplasmin-positive children who had spent the majority of their lives in Region II, which includes California, an endemic area for coccidioidomycosis, failed to react to coccidioidin. One histoplasmin-negative child who had spent 95 per cent of her life in California was also negative to coccidioidin. This numerically scanty evidence corroborates the findings of other studies[21, 22, 25, 46, 48, 51] that cross reactions, while they occasionally occur, are of no real importance in the interpretation of histoplasmin sensitivity in the central part of this country.

Epidemiological Aspects. Present concepts of histoplasmosis have resulted from a critical examination of disease processes that were formerly considered to be almost exclusively of tuberculous origin. Up to 1945 histoplasmosis had been considered a rare and invariably fatal disease. At that time the investigations of Christie and Peterson[10, 11] clearly proved the existence of a widespread mild infection. The pattern of age response is such as one would expect from a widely disseminated infectious

agent. The age at which sensitivity is acquired, the relative absence in young infants, and the progressive rise in prevalence of sensitivity with increasing age indicate that histoplasmosis is not a household contagion but is related to broader and less localized factors.

Additional evidence against person-to-person transmission lies in the results of family studies. In the present investigation all siblings of 123 families with more than one child were tested. Of 71 families with 2 siblings, both siblings were histoplasmin positive in only 5 instances; in no instance did all siblings of families with 3 children show positive reactions. Although there was a slightly greater chance for the older sibling to react to histoplasmin, there was no concentration of reactors in some families and of nonreactors in other families. This is similar to the result obtained in intrafamily studies in Kansas City, Mo., by Ferebee and Furcolow.[52] The reported cases[53, 54] of systemic histoplasmosis in siblings and persons in close contact are probably due to exposure to a common source, as postulated by Beadenkopf and Loosli,[55] rather than to person-to-person transmission.

Although many species of animals have been shown to harbor the fungus, there is no substantial evidence of transmission from animal to animal or animal to man.[56-65] The organism has been isolated from the soil in many areas of the United States,[61, 66-69] including the soil and river water in adjoining Williamson County, Tenn.[70, 71] Residents of a damp environment are more likely to be reactors than those from dry environments,[26, 71-75] dampness apparently being an in vitro growth requirement for *H. capsulatum*.[76] Higher sensitivity rates have been observed in rural than in urban residents.[77, 78] Zeidberg, Dillon, and Gass[13] reported a rise of histoplasmin sensitivity in the summer and early fall, but no significant seasonal change was noted in the current investigation: January-March, 14.5 per cent; April-June, 13.2 per cent; July-September, 13.1 per cent; and October-December, 15.4 per cent.

In the past several years, many epidemics of pulmonary disease of obscure etiology were reported[66-68] but more recently evidence has been presented that the etiologic agent was *H. capsulatum*.[66-68, 79, 80] In most cases there was a history of exposure to dust contaminated with excreta from pigeons, chickens, or bats, which probably contributed factors favorable for the growth of the organism. Ibach, Larsh, and Furcolow[81, 82] isolated the organism from the air of unused chicken houses in Kansas and Missouri, where several individuals engaged in cleaning these houses had developed acute pulmonary histoplasmosis.

Figure 1 illustrates the proximity to a creek and river of the Air Force base where the present investigation was conducted. Eighty-eight per cent of the children tested resided in the housing project, as did 89 per cent of the histoplasmin reactors. The center of the housing project was 5,300 feet from the river and 1,600 feet from the creek, and there were no bodies of water within the project. By criteria of Zeidberg and associates[71] this would be considered a "dry" environment, and a large majority of the total group tested were infants and young children, so that it is unlikely that their range of activity would extend to either the creek or the river. However the soil at the housing project is shallow, slightly acid, and of low permeability and organic content. This is the same as the type I soil of neighboring Williamson County, from which *H. capsulatum* has been isolated repeatedly.[70,71] In addition to soil similarities there is very little difference in the geographic and climatologic characteristics of Williamson and Rutherford Counties; elevation and average annual temperature and rainfall are essentially the same*

RESULTS AND DISCUSSION; TUBERCULOSIS

Although the primary concern of this investigation was with histoplasmosis, the findings related to tuberculin sensitivity are of interest because they reconfirmed the absence of any regionalization of high tuberculin reaction rates to the populace of the lower Mississippi River Basin where the prevalence of pulmonary calcification is greatest. Table 4 reveals that only 17 (2.6 per cent) of the 663 subjects reacted to tuberculin. This contrasts sharply with the 14.0 per cent histoplasmin rate. There was a sex difference in that 12 of 341 males as compared to 5 of 322 females reacted to tuberculin, and prevalence increased with age. The percentage of reactors (2.3 per cent) in the age group below 5 years was identical with that in the age group from 5 to 10 years, but the rate increased to 5.2 per cent in the small number tested who were over 10 years of age.

Table 5 compares the results with those of three previous studies of *native* Tennessee children. It can be seen that there has been a progressive decrease in the prevalence of tuberculin sensitivity in Tennessee children in recent years, reflecting the marked success of case-finding methods and advances in management of the clinical disease.[83,84] Although many surveys of tuberculin sensitivity in native children of different areas have been reported,[83,85-88] no report could be found concerning this problem in a heterogeneous group of children who had lived in all parts of the United States. The high rates in natives of Regions I and II (table 4) can be partly explained on the basis

*Climatological Data, Tenn., Vol. 57, No. 13, U. S. Dept. of Commerce.

of the small number of subjects available for testing from these regions and the fact that two of four reactors from Region I were siblings.

TABLE 4

TUBERCULIN SENSITIVITY, INITIAL TESTS

Classification	Number Tested	Tuberculin Positive	
		No.	%
Total	663	17	2.6
Sex			
Male	341	12	3.5
Female	322	5	1.6
Age			
<1 year	28	1	3.6
1 year	83	2	2.4 } 2.3
2 years	232	5	2.2
5 years	262	6	2.3 } 2.8
>10 years	58	3	5.2
Residence in Region IV			
<1 year	157	5	3.2
1 year	138	5	3.6
2 years	78	0	0.0
3 years	49	0	0.0
4 years	26	0	0.0
>5 years	50	1	
No Record	165	6	3.6
Major Region of Residence			
USA. I	18	2	11.1
II	20	4	20.0
III	50	1	2.0
IV	257	2	0.8
V	91	1	1.1
VI	27	0	0.0
Foreign	35	1	2.9
No Record	165	6	3.6

Sixteen of the 17 tuberculin reactors were studied for signs of disease. By the usual criteria,[89] six children were considered to have healed or inactive primary pulmonary tuberculosis, and six had active pulmonary tuberculosis which healed without complications. Two children had tuberculous cervical lymphadenitis

TABLE 5.

TUBERCULIN SENSITIVITY IN CHILDREN RESIDING IN TENNESSEE

AUTHOR	YEAR		% Positive Reactions In Each Age Group			
			0-5	5-10	10-15	15-20
ARONSON [90]	1933	Rural Tenn. Children	—	36.8	60.8	67.4
GASS [7]	1938	Rural Tenn. Children	—	—	49.0	48.8
CHRISTIE & PETERSON [22]	1946	Middle Tenn. Children	3.1	12.6	24.9	—
CURRENT STUDY	1952-3	Children From All Parts Of U.S. Currently Residing In Tenn.	2.3	2.3	5.2	—

that was cured by surgical and antibiotic therapy. One child with progressive primary pulmonary disease recovered on bed rest and antimicrobic therapy. A 2½-year-old boy was found to have very early tuberculous meningitis; he has remained entirely normal for 30 months after completion of treatment.

SUMMARY

A group of 663 children who had resided in all parts of the United States and abroad and were now living in middle Tennessee were tested for skin sensitivity to histoplasmin and to tuberculin. Of the 663 initially tested, 93 (14 per cent) reacted to histoplasmin and 17 (2.6 per cent) to tuberculin. Only 4 (0.6 per cent) reacted to both antigens. Ten cases of active tuberculosis, including one of early tuberculous meningitis, were discovered by means of this study.

Sensitivity to histoplasmin was rare in infancy and rose with increasing age. There were no sex or seasonal differences. Children who had spent the majority of their lives in states bordering the mid-portions of the Mississippi and Ohio Rivers exhibited the greatest incidence of histoplasmin sensitivity. The likelihood of a positive reaction also increased with duration of residence in this area (middle Tennessee). The sensitivity rates found in this investigation were lower than those reported by others for native children in the east central United States because of the rigid technics of testing and interpretation employed and the fact that many of the subjects had resided mainly in areas where less opportunity for exposure to the infecting organism existed. The findings in this study confirm reports of others that in the central portion of the United States histoplasmosis is a more common cause of pulmonary calcification than is tuberculosis. The results of family studies are evidence against person-to-person transmission of the infection.

Acknowledgment is made of the technical assistance of A/2C Jean Kowalenko.

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DIET AND ATHEROSCLEROSIS

"The ingestion over the years of a diet rich in cholesterol-lipid is an *essential prerequisite* for the development of significant atherosclerosis in a population group. Such a diet may be regarded as an essential 'trigger' for the atherogenic process (viewed as a group phenomenon). Once the trigger is pulled (i. e., once the prequisite diet is ingested), individual differences—endogenous factors (hereditary, constitutional, etc.)—come into play to influence whether the charge is fired (i. e., whether the given person develops significant atherosclerosis)."

—LOUIS N. KATZ, M. D.
in *Annals of Internal Medicine*
p. 933, Nov. 1955

CHARACTERISTICS AND TREATMENT OF MILIARY TUBERCULOSIS

STEPHEN J. BERTÉ, *Lieutenant Colonel, MC, USA*

IN DISCUSSING the pathogenesis of tuberculosis, the difference between the childhood (primary) and adult (reinfection) types of tuberculosis always arises. This, in effect, is the difference between tuberculosis in the tuberculin-negative person and tuberculosis in the tuberculin-positive person. The basis of this difference is the presence or absence of allergy and immunity.

What is allergy and immunity in tuberculosis? In general, the presence of allergy is recognized when a positive tuberculin skin test is observed. Immunity is the acquired or inherent ability of the host to maintain the disease within certain boundaries and to reduce dissemination. It has been observed that the initial cellular response to infection differs in tuberculin-negative and tuberculin-positive patients. Pleural effusion due to tuberculosis does not occur until tuberculin allergy is demonstrated clinically. The early cellular response within the lung of the previously uninfected host is a lobular pneumonia with no tendency to wall off the lesion with a fibrous capsule. Macrophages engulf *Mycobacteria tuberculosis* and transport them to the regional lymph nodes via the lymphatic vessels. In the previously infected host there is a more immediate proliferative response. Macrophages undergo lysis and there is little transportation of organisms via the lymphatics. Necrosis, epithelioid cell proliferation, and fibroblastic response occur earlier and tend to delimit the caseous tubercle within local boundaries. On the one hand, it is theorized that the presence of tuberculin allergy is responsible for pleural effusion, lysis of phagocytes, necrosis, and rapid delimitation of the tubercle. However, some authors suggest that allergy is an incidental factor and that acquired immunity dictates the type of cellular response in tuberculosis. In reinfection tuberculosis, both allergy and immunity are usually present. For the present it is probably wiser to assume that both may contribute in some way to the altered tissue response.

Summary of a lecture given before the Eighth Annual Symposium on Pulmonary Diseases at Fitzsimons Army Hospital, September 1955.

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DISSEMINATION OF TUBERCULOSIS

Theories of dissemination of tuberculosis are based on animal experiments, clinical observations, pathologic examinations, and the anatomic similarity of the lymphatic drainage of the lung in man and the guinea pig. Starting with a droplet infection in the lung, the sequence of evidence that follows may be summarized in the following outline form:*

I. Primary infection in the tuberculin-negative patient

A. When bacilli in a droplet come to rest in a more central portion of the lung:

1. A lobular pneumonia develops.
2. Macrophages engulf bacilli and carry them to hilar and mediastinal nodes via the deep lymphatics.
3. As the pneumonic process enlarges, central caseation may become evident. At the time of caseation there is a lysis of phagocytic cells and infecting organisms. Giant cell formation occurs which, in all probability, is a response to a "foreign body" composed of necrotic cellular debris.

B. Prognosis of an initial infection in a more central portion of the lung drained by the deep lymphatics:

1. Complete resolution leaving a small fibrous residual, or resolution with the development of a Ranke complex.
2. Local progression to an extensive pneumonic process, plus enlargement of hilar and mediastinal nodes.
3. The infection may be maintained within local boundaries and possess viable bacilli within a necrotic focus for months or years. This latent infection may be the cause of subsequent endogenous reinfection.

C. When bacilli in a droplet come to rest in a subpleural location:

1. A pathologic process develops similar to that described in the more centrally located lesions.
2. Few tubercles develop on the surface of the pleura and there is little pleural reaction and no effusion.
3. Bacilli are transported via the superficial lymphatics of the lung to the parasternal and para-aortic nodes. It is remotely possible for organisms to be carried by the superficial lymphatics around the periphery of the lung to the hilum.

D. The prognosis of an initial infection in a subpleural location is basically similar to the more centrally placed lesions with the following exceptions:

1. The lymphatic drainage of infecting organisms is different in that there is no direct communication between the deep and superficial lymphatics of the lung.

*The greater portion of information in the subsequent outline was obtained from publications by Burke[1] and Medlar.[2]

2. Unresolved tuberculous disease may progress and produce pleural effusion with numerous tubercle formations and pleural adhesions. This occurs within two to six weeks at about the time the patient develops a hypersensitivity to tuberculoprotein.

II. Infection in the tuberculin-positive patient

A. The sequence of events after infection occurs in the following chronologic order:

1. A small area of lobular pneumonia occurs.
2. Caseous necrosis develops with the typical pathologic picture of tuberculosis.
3. Liquefaction of caseous contents of the tubercle may occur.
4. The liquefied contents of the tubercle may then be sloughed into the lumen of a communicating bronchus and cause new tuberculous disease in other areas of the lung.

B. Prognosis of the pathologic lesion in the tuberculin-positive patient:

1. In the highly immune person the small area of lobular pneumonia may completely resolve, leaving no residual, or may heal by simple fibrosis.
2. Once caseous necrosis develops, the lesion may remain latent for a long time and eventually heal by fibrosis or it may become liquefied.
3. Once liquefaction develops, sloughing of the lesion into a bronchus nearly always occurs. It is difficult for the body to dispose of a foreign body in the form of inspissated liquid contents of a tuberculous lesion by any other means than sloughing it into a bronchus.

Miliary tuberculosis of the lungs and meninges is the result of hematogenous dissemination of *Myco. tuberculosis*. Following a tuberculous infection in the body, bacilli may enter the blood stream by any of the following routes:

1. A caseous focus may rupture into a pulmonary vein with dissemination via the left auricle and ventricle.

2. Mediastinal, parastinal, or para-aortic nodes may rupture into the venous system or spread into the venous system by way of the thoracic duct and be disseminated via the right auricle and ventricle. A greater proportion of bacilli in the tuberculin-negative patient, and a smaller proportion in the tuberculin-positive patient, may be carried through the pulmonary circulation and be disseminated through the systemic circulation. (Animal experiments suggest that most of the organisms transported to the lungs by the blood are detained in the lung by some yet not understood immune mechanism in the previously immunized animal.)

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3. A secondary, tuberculous, caseous focus in some other organ such as the prostate may rupture into a vein and be carried to the pulmonary circulation.

4. In general, the extent and location of disease following hematogenous dissemination depends upon the number of bacilli disseminated and the immunity of the host.

ROENTGENOGRAPHIC PICTURE

A roentgenogram of the chest revealing diffuse, bilateral, fine miliary shadows is essential to the diagnosis of miliary tuberculosis, which is characterized by miliary "seeding" in both lungs (figs. 1, 2, and 4); however, a diagnosis of miliary tuberculosis should not be accepted simply on the basis of a typical chest roentgenogram. Great caution should be practiced, because there are over 80 primary pulmonary and systemic diseases with pulmonary manifestations that may produce diffuse miliary shadows on the x-ray film. The diagnosis of miliary tuberculosis is suspected from the roentgenogram (figs. 1-4), definitely proved by the isolation of *Myco. tuberculosis,* and (in the absence of positive bacteriologic findings) is presumptively proved by clinically eliminating other possibilities and observing the response to a course of specific chemotherapy.

SIGNS AND SYMPTOMS

There is a great variation in the signs and symptoms. I have seen patients who denied all symptoms and had been brought to the hospital only because a routine roentgenogram had revealed diffuse, miliary shadows in both lungs. At the other extreme, a patient with proven miliary tuberculosis with meningitis was recently admitted to this hospital in profound coma. Despite this variation, the majority of patients admitted to hospitals are well oriented but appear ill and demonstrate chills, fever, anorexia, and malaise. Occasionally the presenting clinical picture may be "flu-like" or "typhoid-like." Rarely, jaundice is seen with involvement of the liver or in association with secondary hemolytic anemia. An analysis of patients with miliary tuberculosis at this hospital within the past three years revealed that (1) 48 per cent complained of cough; (2) 64 per cent exhibited auscultatory findings in the lungs, and of these findings, 80 per cent consisted of bilateral, inspiratory rales; and (3) extrapulmonary tuberculosis was detected in 92 per cent of cases, and of these, 52 per cent consisted of tuberculous meningitis as a coexistent form of extrapulmonary tuberculosis.

LABORATORY EXAMINATIONS

The complete "routine" blood cell count and sedimentation rate is of no diagnostic aid. The blood count may be of aid in

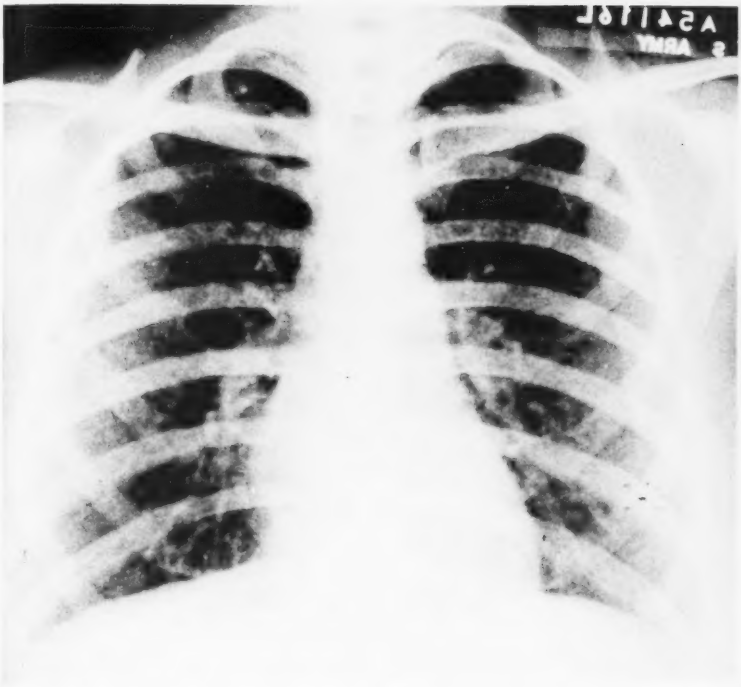

Figure 1. Extremely fine, diffuse miliary nodulations seen in the lung of a patient with miliary pulmonary and meningeal tuberculosis.

suspecting a secondary anemia, hemolytic anemia, or a pancytopenia with extensive bone marrow tuberculosis.

A positive tuberculin skin test adds great support to the diagnosis before the specific micro-organism is isolated. However, a negative tuberculin skin test may be found under the following conditions:

1. In a first infection with early hematogenous dissemination.

2. Tuberculin anergy may occur in the severely ill person.

3. Allergy to tuberculoprotein may be lost many years after an original infection and may not reappear until a short time after reinfection tuberculosis is well established.

4. Improperly prepared or administered tuberculin may give a false negative test.

Serologic studies for such fungal diseases as histoplasmosis, coccidioidomycosis, and blastomycosis are available and should

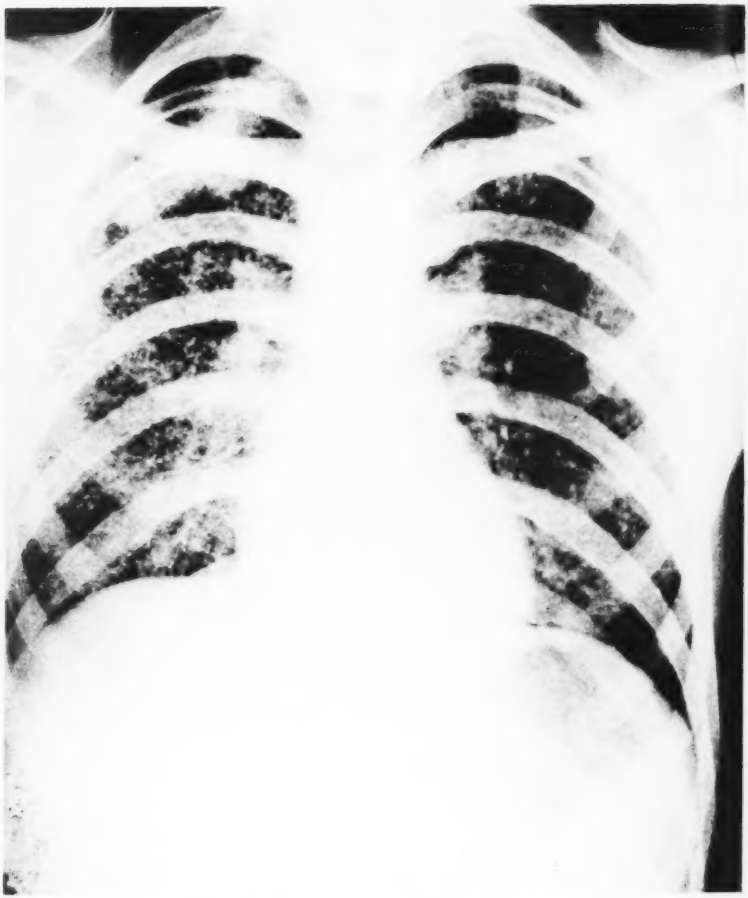

Figure 2. Moderately coarse, diffuse, miliary nodulations are present. The patient received 28 months of combined drug therapy.

be obtained. These latter diseases may completely mimic the clinical and roentgenographic picture of miliary tuberculosis.

Various body fluids that should routinely be examined for the presence of *Myco. tuberculosis* are:

1. Sputum—concentrated smear and culture.

2. Gastric fluids—culture. (Examination by direct smear may add confusion to the diagnosis because of the frequent finding of nonpathogenic acid-fast bacilli.)

3. Urine—concentrated smear and culture of a 24-hour specimen.

Figure 3. Same patient as in figure 2 after the roentgenographic findings returned to normal. Miliary nodulations had disappeared after four months of chemotherapy.

4. Cerebrospinal fluid—smear and culture. (The finding of acid-fast staining bacilli in the protein pedicle of spinal fluid may be accepted as proof of tuberculous meningitis. The chance finding of acid-fast staining organisms, other than *Myco. tuberculosis,* in this location is quite unlikely.)

Cultures for *Myco. tuberculosis* are preferred to guinea pig inoculation when highly qualified laboratory personnel are available. However, guinea pig inoculation of suspected material may be preferred when the laboratory is not equipped to do a detailed research type of analysis of micro-organisms.

Some authors,[3-7] have obtained gratifying results in the diagnosis and treatment of miliary tuberculosis with the aid of bone marrow and liver biopsies. This practice has not been evaluated at this hospital.

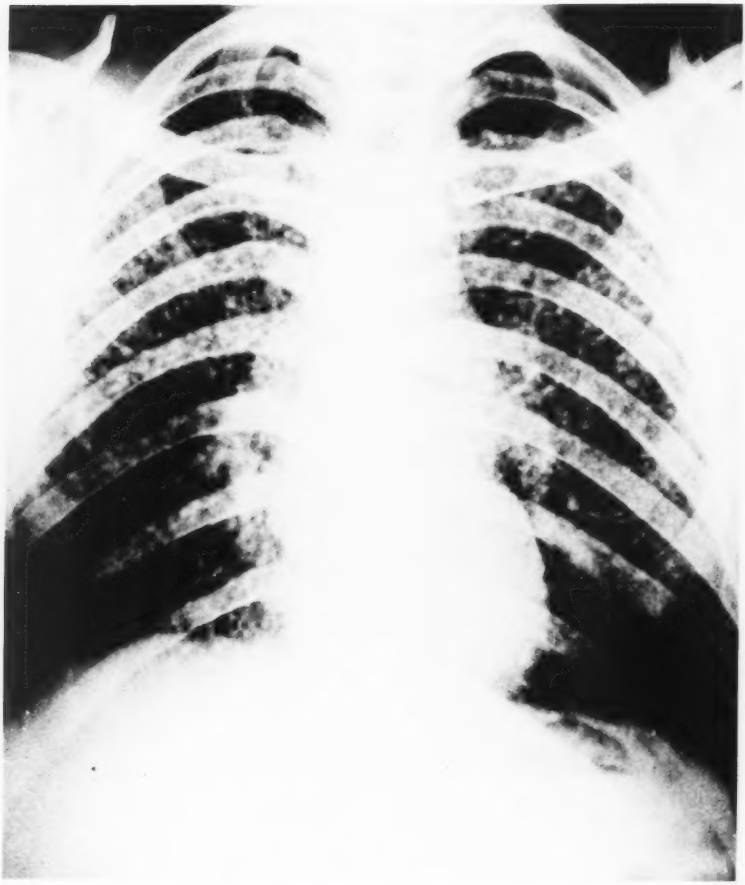

Figure 4. Coarse, conglomeratic, miliary nodulations with numerous circular densities or "pseudocavities" in the lung of a patient with miliary pulmonary tuberculosis.

A review of cases at this hospital indicates that, of patients with miliary tuberculosis admitted within the last three years, 64 per cent of cases were proved by bacteriologic means. The remainder were diagnosed presumptively by excluding other possible diagnoses and observing a typical response to antituberculous chemotherapy.

PATHOLOGY

The pulmonary lesions of miliary tuberculosis are produced when many bacteria are disseminated via the pulmonary artery and are lodged at multiple sites throughout the lung parenchyma. As a result, many minute areas of lobular pneumonia are produced. These areas then form a noncaseating tubercle which heals by complete fibrosis under the influence of chemotherapy. Occasionally this healing process can result in diffuse pulmonary fibrosis with some degree of alveolar-capillary block to the diffusion of gases. Medlar described an occasional form of miliary tuberculosis called "acute caseating miliary tuberculosis" in which caseation is a prominent component of each miliary tubercle. Such a condition may be associated with an untreated, overwhelming infection in which many bacilli are deposited at each site. A high tuberculoprotein sensitivity and, perhaps, a temporary reduction in immunity may be contributing factors.

TREATMENT

Miliary tuberculosis was virtually 100 per cent fatal before the advent of specific chemotherapy. "Healed" calcified miliary tuberculosis has been described, but this, in all probability, represents miliary calcifications of histoplasmosis or aspergillosis.

An understanding of the treatment of miliary tuberculosis can best be appreciated by reviewing the "evolution" of chemotherapy since 1946. At first, streptomycin sulfate (SM) was administered alone for short periods of time. Smith[8] reviewed one hundred cases treated daily with SM for two and one-half to four and one-half months. The survival two and one-half years after treatment was miliary tuberculosis alone, 47 per cent; miliary tuberculosis with meningitis, 3 per cent; and meningitis alone, 16 per cent. Other reports[9,10] revealed similar survival data with variations of plus or minus 5 per cent.

Des Autels and Pfuetze[10] found the increase in survival when the daily dose of SM was combined with para-aminosalicylic acid (PAS) for a period of at least 12 months, as follows: miliary tuberculosis alone, 83.3 per cent; miliary tuberculosis with meningitis, 71.4 per cent; and meningitis alone, 59.3 per cent. Debre[11] reviewed the results in 170 children with miliary and meningeal tuberculosis treated for from 8 to 13 months and found similar survival rates following a five-year follow-up period.

At the Thirteenth Veterans Administration-Army-Navy Conference on the Chemotherapy of Tuberculosis[12] at St. Louis, Mo., in February 1954, the great significance of isoniazid (isonicotinic acid hydrazide, INH) in the treatment of miliary tuberculosis was emphasized. In a review of several hundred cases it

was shown that 40 per cent of patients with miliary tuberculosis treated with SM alone developed meningitis within one year of the beginning of therapy. In patients treated with SM and PAS, only 14 per cent developed meningitis within one year. When INH was combined with SM and PAS, no patients developed meningitis during a six months' follow-up period. The over-all survival in miliary tuberculosis with meningitis was twice as great in SM-PAS-INH treated cases as in SM-PAS treated cases in a 6- to 18-month follow-up study.

Data extracted from the consolidated tables of the Fourteenth Veterans Administration-Army-Navy Conference on the Chemotherapy of Tuberculosis[13] given at Atlanta, Ga., February 1955, summarized the results of chemotherapy to date. Table 1 illustrates this in a review of 728 cases (140 of which were treated with INH).

TABLE 1

Type of tuberculosis	Drug regimen	Per cent of survival after 18 months
Miliary alone	SM	64
	SM–PAS	81
	SM–INH, or SM–INH–PAS	95
Miliary with meningitis	SM	16
	SM–PAS	41
	SM–INH, or SM–INH–PAS	80
Meningitis alone	SM	25
	SM–PAS	52
	SM–INH, or SM–INH–PAS	78

(Note: In the Sm– and SM–PAS-treated groups above, survival at 18 months was similar to survival data at five years.)

At this hospital, triple drug therapy using SM-INH-PAS gave the best results in miliary tuberculosis. Roentgenograms of patients treated with SM-INH-PAS showed clearing of miliary shadows in an average of 6.8 months (fig. 3). Roentgenograms of patients treated with varying combinations of SM, PAS, and viomycin sulfate showed clearing of miliary shadows in an average of 10.6 months. No relapses occurred in patients with miliary tuberculosis treated for at least 18 months. Relapses occurred in patients with tuberculous meningitis treated for less than 2 years.

The treatment of miliary tuberculosis with INH alone has been under investigation. The theory of such treatment is based upon the character of the minute, noncaseating granuloma. It is thought

that INH can penetrate this lesion, eliminate all multiplying organisms, and promote complete healing. The drug is continued for a long time because some nonmultiplying, intracellular organisms may maintain their viability and institute regrowth with early discontinuance of chemotherapy. For the present, it must be assumed that INH alone is not proved to be equal to the combined chemotherapeutic methods.

RECOMMENDED DRUG THERAPY

Upon the basis of the foregoing observation, the following recommendations regarding drugs are made in connection with the treatment of miliary pulmonary and meningeal tuberculosis:

1. Use triple drug therapy consisting of SM-INH-PAS:

   a. Give 1 gram of SM daily, for a three-month period. If a decided response has been observed, SM may then be continued in doses of 1 gram every third day.

   b. Give INH in a dosage of 10 to 16 milligrams per kilogram of body weight per day. (Pyridoxine, 100 milligrams daily, should be administered with INH to reduce neurologic complications.)

   c. Give PAS in a dosage of 12 grams daily divided into three or four portions.

2. When the patient is in coma or is vomiting, INH may be given in the same dosage by intramuscular injection and PAS can be given intravenously, using 15 grams of the lyophylized crystals.

3. Drug therapy should be continued for at least 18 months in miliary tuberculosis without meningitis and at least 24 months in tuberculous meningitis. The roentgenographic findings or spinal fluid chemical studies should have been relatively normal for more than six months before chemotherapy is discontinued. At this hospital, we have recommended for prophylaxis at least 24 months of chemotherapy for miliary tuberculosis without meningitis and at least 30 months of chemotherapy for tuberculous meningitis. A few patients with meningitis with more severe manifestations have received three years of chemotherapy. Isoniazid may be reduced to about 5 milligrams per kilogram of body weight per day when the patient is allowed to go home. Such a reduction in the dosage of INH should probably not be considered until at least 18 months of chemotherapy have been completed.

4. SM intrathecally is not recommended in tuberculous meningitis; and, although we have not used cortisone and corticotropin at this hospital, recent reports[14,15] suggest that their early trial may warrant consideration.

SUMMARY

Miliary tuberculosis is the result of hematogenous dissemination of *Mycobacteria tuberculosis*. Roentgenograms of the chest reveal small diffuse nodulations in all areas of both lungs. The infecting organism enters the venous circulation via the thoracic duct, rupture of a caseous lymph node into an adjacent vein, or from a distant source such as the prostate. There are differences in the dissemination of *Myco. tuberculosis* in primary and reinfection tuberculosis. In the initially infected host, bacilli are transported more rapidly to the regional lymph nodes via the deep and superficial lymphatics of the lung. The majority of disseminated organisms in initial infections in tuberculin-negative hosts pass through the pulmonary circulation and may lodge in the meninges or other organs. In reinfection tuberculosis, it is believed that most of the organisms are "trapped" in the lung and a much smaller per cent pass through to the systemic circulation.

Before the era of modern antituberculous chemotherapy, miliary tuberculosis, with or without meningeal tuberculosis, was virtually fatal, whereas present-day *survival* rates are 95 per cent in miliary pulmonary tuberculosis and about 80 per cent when tuberculous meningitis is present. Original drug therapy for miliary tuberculosis consisted of short courses of streptomycin sulfate (SM) alone. Later, SM was given for longer periods in combination with para-aminosalicylic acid (PAS). The best results are observed when isoniazid (isonicotinic acid hydrazide, INH) is added to SM and PAS, and all three are given for prolonged periods.

Presently recommended drug therapy is as follows: SM in a dose of 1 gram daily for three months, followed by 1 gram two or three times weekly; PAS in three or four divided doses totaling 12 grams daily; and INH in a dose of from 10 to 16 milligrams per kilogram of body weight per day. Pyridoxine (100 milligrams daily) should be administered with large amounts of INH to reduce neurologic complications. The total duration of chemotherapy should extend for 18 to 24 months in miliary tuberculosis alone, and for 24 to 30 months in tuberculous meningitis. Intrathecal therapy of any kind is not believed to be of any proven value.

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"On 1 January 1953, there were 13,342 physicians in the military services, and on 1 January 1955 there were 10,749. Just what do these 10,749 physicians do? First there are approximately 2,900,000 servicemen and then, in addition, about 2,200,000 dependents, and in various foreign countries 29,000 civilian employees with dependents who look to the military for care, which is a potential load of about 5,000,000 people to be cared for by doctors and dentists in the Armed Forces."

—FRANK B. BERRY, M. D.
in *Journal of the Medical Association of Georgia,* p. 569, Dec. 1955

THERAPY OF NONGONOCOCCAL URETHRITIS

Comparison of Oral and Intramuscular Terramycin

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NONSPECIFIC or nongonococcal urethritis is a disease of young, sexually active males, and as such is of importance in the medical care of military personnel.[1-5] Its officially reported incidence in the military is small. The outpatient study, evaluation, and treatment of these cases is prolonged and expensive; the assurance of a "cure" cannot be optimistically given. In spite of a considerable amount of clinical research on this disease,[6-11] its etiology and epidemiology have yet to be definitely established; it is believed by most that its cause is related to bacterial or viral infection of the lower genito-urinary tract, and that it is transmitted from the female carrier to the male by sexual intercourse in much the same fashion as is assumed for some other infections.

Treatment with various antibiotics[12-15] has been partially successful, at least in relatively short-term follow-ups, and would seem to substantiate the belief that bacteria certainly play an important role, although, perhaps, do not explain the entire picture. It is a disease, however, that tends to recur even after initial success with antibiotic treatment. There is much dispute as to what antibiotic or combination of antibiotics is most efficacious in bringing about a cure, or at least a clinical remission. Terramycin (brand of oxytetracycline) in the intramuscular form was recently made available to us. The purpose of this study was to compare the clinical and bacteriologic results of treatment with this form of Terramycin with some other commonly used drugs.

MATERIAL AND METHODS

Subjects consisted of personnel who appeared at the infirmary with a persistent urethral discharge negative for gonococcus on direct smear on three successive days, and negative for *Neisseria gonorrhoeae* on culture. At the time of the third smear, the urethral exudate was cultured; the organisms present were identified and routine sensitivity tests performed to penicillin, strepto-

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mycin sulfate, Aureomycin (brand of chlortetracycline hydrochloride), Terramycin, and Chloromycetin (brand of chloramphenicol) by the disk method. After the above procedures were performed the patient was interviewed by a physician who took a history and performed a physical examination that included palpation of the prostate gland. Material was expressed by prostatic massage for smear, routine and *N. gonorrhoeae* culture, and routine sensitivity tests. All cultures were obtained by swab from the urethral meatus and transported to the laboratory in non-nutrient media that were later streaked out on both blood agar and chocolate agar plates under CO_2 tension. There were 57 patients included in this study.

Five different methods of treatment were prescribed, each assigned in rotation without regard to the organisms isolated or to their sensitivities, and regardless of the gross nature or severity of the discharge. The five treatment groups evolved were as follows:

1. *Control group.* These patients were given gelatin capsules containing sucrose and were instructed to take one every six hours for five days.

2. *Group on Terramycin intramuscularly.* These patients were given 100 mg of Terramycin as the crystalline hydrochloride, 100 mg of magnesium chloride hexahydriate, and 2 per cent (w/v) procaine hydrochloride in 2.1 ml of sterile water. This was injected into the gluteus muscle once daily for two days.

3. *Group on Terramycin orally.* This group was given 250 mg of Terramycin every six hours for five days.

4. *Group on penicillin.* This group was given 360 mg (600,000 units) of procaine penicillin G intramuscularly once daily for five days.

5. *Group on sulfadiazine.* This group was given 4 grams of sulfadiazine initially, then 1 gram every six hours for five days, and 2 grams of sodium bicarbonate every six hours.

In all instances the patients were given the following additional advice: (1) no sexual intercourse for one month, (2) no alcoholic beverages for one month, (3) no more than three cups of coffee per day, (4) no "milking down" of the urethra at any time, and (5) limited exercise.

Patients were seen one week after the start of therapy, at which time the clinical response was evaluated. A rectal examination was performed; the prostate gland was massaged, and any material expressed was again cultured and sensitivity tests performed; any urethral exudate was also cultured. Patients were seen again one month following the start of therapy, at which time the same procedures were performed.

RESULTS

Incubation Period. We believe that no valid, clear-cut "incubation" period can be established for this disease, at least from our data and experience. It is true that in some cases a patient, previously free of symptoms, has a single sexual contact and several days later (often stated to be about 10 to 14 days) develops the symptoms of urethritis. However, because of this disease's variable nature and its tendency to recur after a quiescent period, a definite incubation period is difficult to establish. In many cases there have been multiple sexual contacts with the same or different women; other times the discharge appears to be a residuum of a successfully treated gonococcal urethritis. Frequently the patient has had symptoms intermittently or continuously for many weeks or months, varying with his physical activity, amount of fatigue, alcoholic intake, sexual activity, and various unknown factors.

Our incubation period, actually representing the period between the last contact and the appearance of symptoms and therefore not an incubation period in the bacteriologic sense, varied from 1 day to 2 months, and in 6 cases it could not definitely be established. Of our 57 cases, 10 occurred 4 days or less after contact, and 28 (about 50 per cent) occurred 5 to 15 days after the last contact. The median was 7 days, and the average was 11 days.

Age and Race. There were 41 white patients, 3 Negro, 3 Oriental, and 10 whose race was not recorded. These results seemed to reflect those of other investigators who have found a high prevalence of this disease among those of the Caucasian race, as opposed to gonorrhea, which seems to have a somewhat higher prevalence among those of the Negro race. The patients' ages varied from 19 to 39 years; the majority (33) were between 19 and 23 years inclusive, with the prevalence falling off rapidly after that. Our population, of course, was selective and only reflected the relatively young age of the personnel stationed at our base.

Previous Venereal History. Thirty-seven per cent of the patients had previously had gonorrhea; two patients had had four prior episodes and two others, three episodes. All had been adequately and successfully treated with penicillin. In a few cases the nonspecific urethritis seemed to be in some way related to this gonococcal infection in that their nonspecific discharge continued after their gonococcal urethritis had been cured. It is possible that both infections had been contracted at the same time, and the penicillin affected the gonococcus but not the other organisms. Another possibility is that the continued discharge may only represent an irritative factor or some phenomenon as yet poorly understood. In 53 per cent of our cases there had been one or more previous episodes of nonspecific urethral discharge, sometimes

lasting many months. Two patients had a history of chancroid, but none had had syphilis. Incidentally, primary syphilis was an extremely rare condition at our installation.

Precipitating and Aggravating Factors. These are varied, and many are unknown. In many of our cases (60 per cent) there was a history of heavy alcoholic intake at the time of the last sexual exposure. This does not necessarily implicate alcohol as being a factor in acquiring the disease; however, in many patients alcohol seemed to reactivate a relatively inactive or latent case. Strenuous physical activity sometimes increased an already established discharge or made manifest an infection that had been in remission, and may account for the term "strain" used by many patients in association with this disease. Also, violent "milking down" of the urethra by overly concerned patients frequently prolonged the course of the disease.

Prophylaxis. Only 10 per cent of our patients used a condom, and this brings up the question of whether or not its more universal use would prevent nonspecific urethritis. (It has been our experience that the use of a condom definitely reduces the chances of contracting gonorrheal urethritis.) About 50 per cent of the patients stated that they washed and urinated within five minutes after intercourse; thus it would appear that this is not an effective method of control. No patients reported the use of intra-urethral medication or self-administered antibiotic medication.

Symptoms. The most prominent symptom of this disease, urethral discharge, was used as the major criterion for the evaluation of therapy. All of the patients had this symptom to a varying degree—from an intermittent, mild, clear, morning discharge to a continuous, more profuse, purulent discharge. In most cases the discharge was cloudy and milky in nature, occurring usually upon arising in the morning and after urinating. In many instances the discharge was grossly purulent, but rarely, if ever, was it as profuse as in most cases of gonorrhea, and rarely did it have the rather typical pale yellow color of the gonococcal urethral exudate. Many patients with nongonococcal urethritis were examined but were not included in this series because their discharge was so mild and intermittent that it offered little basis for later evaluation of therapy. Undoubtedly many of these had in reality a traumatic urethritis produced by excessive sexual activity or were overanxious individuals who "milked down" the urethra; in other patients the discharge represented only a slight increase in the normal urethral mucus. In several other patients it cleared spontaneously during the process of obtaining preliminary smears; these, therefore, were not included in the series.

Burning on urination occurred in about 50 per cent of our patients and seemed to occur more often than with gonococcal

urethritis. An itching feeling inside the penis or glans was also frequently mentioned. Other symptoms mentioned rather infrequently were low back discomfort or pain; groin, testicular, or perineal discomfort; and frequency of urination. These latter symptoms more often seemed to be associated with the more apparent degrees of prostatic involvement.

Physical Findings. Aside from the urethral discharge, the external examination was usually negative for significant lesions. The most striking finding was prostatic involvement in about 70 per cent of our patients. This was usually manifested by an enlargement of from half to once again normal size, as well as by a definite boggy or spongy feeling distinctly different from that of the firm, normal, young male gland. Tenderness was infrequent, and when present it was usually mild. In nearly all of these patients several drops of cloudy fluid could easily be expressed on prostatic massage. In a very few patients the prostate was definitely tender and somewhat nodular, and these patients obviously had a well-developed prostatitis. Perhaps this disease should more correctly be called a "prostato-urethritis." The prostatic involvement may explain the frequent difficulty in successfully treating this disease. We found no consistent correlation between the clinical results of treatment and change in the size and consistency of the prostate gland; in many patients with a clinical cure an enlarged, boggy prostate persisted, whereas in some with clinical failures the gland seemed to revert to normal. However, this aspect of our study was poorly controlled because we did not do prostatic examinations on a control group of normal individuals.

Bacteriologic Data. In table 1 are listed the various bacteria cultured from both urethral and prostatic exudates before and after treatment. There were 103 separate prostatic and urethral cultures before treatment and 37 after treatment. The lesser number after treatment reflects the fact that in the successful cases it was frequently impossible to obtain urethral and/or prostatic exudates.

It is seen that the hemolytic staphylococcus was by far the most commonly isolated organism, occurring in from about 70 to over 90 per cent of the cultures. The flora, however, was mixed in most cases, *Corynebacterium,* nonhemolytic staphylococcus, and streptococci also occurring frequently. It appeared that treatment had little effect on the type of flora or their relative abundance. Sterile exudates were so infrequent in our series as to be considered rare; it is doubtful that one can really sterilize the lower genitourinary tract. Our cultures, even though they showed little difference in flora before and after treatment, gave no index of the number of bacteria or their virulence—both important points to consider in any infection. Thus, the antibiotics could materially affect

the degree of infection and yet their activity not be reflected in our cultures. The fact that hemolytic staphylococcus, a known pathogenic organism, occurred so commonly, plus the fact that our results and those of other investigators have usually been superior with those antibiotics highly effective against hemolytic staphylococcus, would lead one to conclude that this organism must play some role in causing this disease.

Table 2 gives the results of the in vitro resistance studies of eight major isolated organisms. Of the five antibiotics tested, only penicillin and Terramycin were used in our study. It is evident that for hemolytic staphylococcus, penicillin is the most ineffective drug (37 per cent sensitive), and this is in agreement with most other observers. Streptomycin sulfate was 85 per cent effective and this perhaps explains recent reports of good results with combinations of penicillin and streptomycin sulfate. Hemolytic staphylococci were sensitive to Chloromycetin in almost 100 per cent of the cases, and perhaps on this basis this drug would deserve a more extensive trial in the treatment of this disease. Probably the reason for its infrequent use (and thus its effectiveness) reflects the medical profession's reluctance to use routinely a potentially toxic drug in a usually benign, self-limited disease subject to spontaneous remission. We have not hesitated to use it on occasions where all other drugs failed and the symptoms were fairly prominent, and we have frequently, though not uniformly, realized good results. Both Terramycin and Aureomycin seemed to be fairly active against hemolytic staphylococcus, much more so than penicillin but less so than streptomycin and Chloromycetin.

Aside from its poor activity against the hemolytic staphylococcus, penicillin appeared to be fairly active against other staphylococci and streptococci, and against *Streptococcus pyogenes* it continued to be superior to all others. Both Terramycin and Aureomycin, although not being the most potent drug with any one single organism, appeared to be highly effective against practically all of them.

An attempt was made to determine if there was any change in the sensitivities in the organisms after treatment (table 3). In the case of penicillin there appeared to be a decrease in the sensitivity of the organisms isolated after treatment. In the case of Terramycin and Aureomycin there was a decreased sensitivity with staphylococci; no increase in resistance to Chloromycetin was noted.

Results of Therapy. Table 4 summarizes the results of our series. A patient was considered cured if there was prompt clearing of the discharge or if it cleared within two weeks after the start of treatment and did not return. A patient was considered

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TABLE 1. *Organisms isolated and frequency of their occurrence*

Organism	Cultures with organism			
	Before treatment		After treatment	
	No.	%	No.	%
Hemolytic staphylococcus				
Urethra	42	68.8	11	91.7
Prostate	39	92.8	20	80.0
Nonhemolytic staphylococcus				
Urethra	20	32.8	4	33.3
Prostate	9	21.4	9	36.0
Nonhemolytic streptococcus				
Urethra	7	11.5	3	25.0
Prostate	7	16.6	2	8.0
Streptococcus viridans				
Urethra	10	16.4	1	8.3
Prostate	9	21.4	2	8.0
Streptococcus pyogenes				
Urethra	3	4.9	1	8.3
Prostate	0	0	2	8.0
Corynebacterium				
Urethra	31	50.8	8	66.6
Prostate	22	52.2	15	60.0
Micrococcus				
Urethra	2	3.3	0	0
Prostate	2	4.7	0	0
Hemophilus influenzae				
Urethra	5	8.2	0	0
Prostate	2	4.7	0	0
Bacterium alkaligenes				
Urethra	1	1.6	1	8.3
Prostate	2	4.7	1	4.0
Bacillus subtilis				
Urethra	5	8.2	0	0
Prostate	0	0	2	8.0
Paracolobactrum aerogenoides				
Urethra	1	1.6	0	0
Prostate	0	0	0	0
Achromobacter				
Urethra	1	1.6	0	0
Prostate	0	0	0	0
Proteus morganii				
Urethra	1	1 .6	0	0
Prostate	0	0	0	0
Hemophilus canis				
Urethra	1	1.6	0	0
Prostate	0	0	0	0

TABLE 1. *Organisms isolated and frequency of their occurrence*—Continued

Organism		Cultures with organism			
		Before treatment		After treatment	
		No.	%	No.	%
Aerobacter aerogenes					
	Urethra	1	1.6	0	0
	Prostate	2	4.7	0	0
Neisseria sicca					
	Urethra	0	0	0	0
	Prostate	0	0	1	4.0
No growth					
	Urethra	3	4.9	0	0
	Prostate	0	0	1	4.0
Unsuitable					
	Urethra	1	1.6	0	0
	Prostate	0	0	1	4.0
Total	Urethra	61	100.0	12	100.0
	Prostate	42	100.0	25	100.0

improved if the discharge, though not disappearing completely, became scant and clear. A patient with a discharge that cleared promptly but later returned after another sexual exposure or episode of heavy drinking was also considered improved. Therapy of patients in whom the discharge continued essentially unchanged was considered to be a failure. It is seen from the results that no method gave a striking rate of cure. However, it is evident that sulfadiazine and Terramycin given orally were superior in that practically all patients receiving either of these two drugs were helped to some degree, being classified as either improved or cured. Terramycin intramuscularly and penicillin seemed somewhat less effective, and the poorest results were obtained in the control series.

No adverse local reactions, aside from local discomfort that lasted for a short while after the injection, were encountered from Terramycin given intramuscularly. It is apparent that the intramuscular administration of Terramycin in the dosage and schedule used in our study was not as efficacious as were some of the other methods of treatment. However, this by no means indicated that it is ineffective, as a higher dosage and/or more prolonged treatment might prove it to be at least the equal of oral administration. The dosage used in our study was relatively small, and since this drug is bacteriostatic rather than bacteriocidal in effect, it is not surprising that it was not more effective in a disease, such as this one, that may run a subacute or chronic course.

TABLE 2. *In vitro sensitivities in cultures of eight major isolated organisms*

Organism	Penicillin*		Terramycin*		Aureomycin		Chloromycetin		Streptomycin	
	No.	%	No.	%	No.	%	No.	%	No.	%
Hemolytic staphylococcus										
Sensitive	37	36.6	62	62.0	59	58.4	100	99.0	86	85.1
Resistant	64	63.4	38	38.0	42	41.6	1	1.0	15	14.9
Nonhemolytic staphylococcus										
Sensitive	25	65.8	27	72.9	28	73.7	35	92.1	36	94.7
Resistant	13	33.2	10	27.1	10	26.3	3	7.9	2	5.3
Corynebacterium										
Sensitive	55	90.1	57	95.0	57	93.4	61	100.0	58	95.0
Resistant	6	9.9	3	5.0	4	6.6	0	0.0	3	5.0
Streptococcus viridans										
Sensitive	17	80.9	19	90.5	20	95.2	20	95.2	6	28.6
Resistant	4	19.1	2	9.5	1	4.8	1	4.8	15	71.4
Nonhemolytic streptococcus										
Sensitive	13	86.6	10	66.6	10	66.6	14	93.3	5	33.4
Resistant	2	13.4	5	33.4	5	33.4	1	6.7	10	66.6
Hemophilus influenzae										
Sensitive	4	57.1	6	85.7	7	100.0	7	100.0	7	100.0
Resistant	3	42.9	1	14.3	0	0.0	0	0.0	0	0.0
Bacterium alkaligenes										
Sensitive	2	40.0	3	60.0	3	60.0	1	20.0	4	80.0
Resistant	3	60.0	2	40.0	2	40.0	4	80.0	1	20.0
Streptococcus pyogenes										
Sensitive	7	100.0	7	100.0	6	85.7	6	85.7	0	0.0
Resistant	0	0.0	0	0.0	1	14.3	1	14.3	7	100.0

*Antibiotics used in this study

	Resistant	0	0.0	0	0.0	1	14.3	1	14.3	7	100.0

*Antibiotics used in this study

TABLE 3. *Sensitivity studies of four major organisms before and after treatment*

Organism and response to treatment		Penicillin*		Terramycin*		Aureomycin		Chloromycetin		Streptomycin	
		No.	%	No.	%	No.	%	No.	%	No.	%
Hemolytic staphylococcus											
Before	Sensitive	28	37.8	49	66.2	45	60.8	73	98.6	64	86.4
	Resistant	46	62.2	25	33.8	29	39.2	1	1.4	10	13.6
After	Sensitive	8	29.6	13	50.0	14	51.4	27	100	22	81.5
	Resistant	19	70.4	13	50.0	13	48.6	0	0	5	18.5
Nonhemolytic staphylococcus											
Before	Sensitive	19	73.1	21	80.8	20	76.9	24	92.3	26	100
	Resistant	7	26.9	5	19.2	6	23.1	2	7.7	0	0
After	Sensitive	6	50.9	5	50.0	7	63.6	11	100	9	81.8
	Resistant	5	49.1	5	50.0	4	36.4	0	0	2	18.2
Streptococcus viridans											
Before	Sensitive	15	83.6	16	88.8	17	94.4	18	100	5	27.8
	Resistant	3	16.7	2	11.2	1	5.6	0	0	13	72.2
After	Sensitive	2	66.6	3	100	3	100	2	66.6	1	33.4
	Resistant	1	33.4	0	0	0	0	1	33.4	2	66.6
Nonhemolytic streptococcus											
Before	Sensitive	8	88.8	5	55.5	5	55.5	8	88.8	3	33.4
	Resistant	1	11.2	4	44.5	4	44.5	1	11.2	6	66.6
After	Sensitive	4	80.2	4	80.0	4	80.0	5	100	1	20.0
	Resistant	1	20.0	1	20.0	1	20.0	0	0	4	80.0

*Antibiotics used in this study

TABLE 4. *Results of therapy*

Group	Cured		Improved		Unimproved		Total
	No.	%	No.	%	No.	%	
Control	5	41.7	3	25.0	4	33.3	12
Treated with penicillin	5	41.7	5	41.7	2	16.6	12
Treated with sulfadiazine	5	45.5	6	54.5	0	0.0	11
Treated with Terramycin intramuscularly	5	41.7	5	41.7	2	16.6	12
Treated with Terramycin orally	6	60.0	3	30.0	1	10.0	10
Total	26	45.6	22	38.6	9	15.8	57

DISCUSSION

Systemic forms of effective medications given orally are usually reserved for treating those patients with serious infections who for some reason cannot take the oral form. This is as it should be, for undeniable in most cases oral administration is easier and more pleasant, is free from pain, and avoids complications (sterile abscess, local tissue reactions, hematomas, acute reactions to the drug, et cetera) that can arise with any injected medication. In addition, this form of parenteral Terramycin contains procaine hydrochloride and therefore has the added risk, though small, of sensitivity to it. However, it was felt that if the intramuscular form showed a clear-cut superiority to the oral form by being as effective in a relatively low dosage and with only very few injections, the disadvantages would be outweighed. The advantages of the intramuscular form would be: (1) the low cost of two to four injections of 100 mg each, as opposed to the higher dosage and more prolonged period of administration with the oral form; (2) more positive control of the patient because the medication is not self-administered, thus avoiding irregular administration by some patients and the wasting of medication; (3) avoidance of gastro-intestinal reactions associated with oral medications; and (4) more certain absorption of the medication. In the dosage and schedule of administration used in our study, the intramuscular form of Terramycin appeared to be slightly inferior to the oral form, and thus justification for its use in this disease does not appear warranted on the basis of our small series. We are currently continuing to enlarge our series of patients receiving this medication and prolonging the period of administration.

Sulfadiazine appears to be a very effective drug. If administered with the proper precautions, it is relatively safe; in those patients engaged in active field work where dehydration may come into play, it would probably be better to avoid it, substituting oral administration of a broad-spectrum antibiotic such as Terramycin, Aureomycin, or Chloromycetin. One might speculate that its effectiveness may be related to its relatively infrequent use in the usual gram-positive 'coccal infections. Unfortunately we were not able to do sensitivity studies with this drug.

Our bacteriologic data show a rather high incidence of hemolytic staphylococcus in the urethral and prostatic exudates, higher than reported by most other investigators. This fact, coupled with the observation that the most effective antibiotics and chemotherapeutic agents in this disease are those known to be active against the staphylococcus, leaves one with the distinct impression that these organisms play an important role in its cause.

Although sensitivity studies on isolated organisms were performed, they were not used as a guide in therapy during the period of observation. In those cases that were considered treatment failures, we resorted to the sensitivity studies in an attempt to choose a more effective antibiotic. It is fully realized that in vitro sensitivities of an organism to a particular antibiotic do not always reflect the organism's response to actual treatment of a patient. We feel it is wise to perform cultures and sensitivity tests of the urethral and/or prostatic exudates when the patient is first seen; these can then be used as a guide in the selection of the best antibiotic for initial therapy. If it is impractical to wait for the results of these studies before initiating treatment, the patient may first be given one of the sulfa drugs or one of the broad-spectrum antibiotics, and if response is poor the previously obtained laboratory results can be used in selecting a more potent drug. By such a procedure we feel a higher cure rate can be obtained. In those instances where the population of the base is relatively stable, with few transients being treated, little or no harm is done by waiting for about five days for the results of culture and sensitivity studies. Of course, those patients with an acute, severe prostatitis or seminal vesiculitis should receive immediate treatment.

SUMMARY

The effectiveness of the intramuscular form of Terramycin as compared with some other standard forms of treatment in nonspecific urethritis was studied in 57 patients. Ninety-five percent of the patients receiving either sulfadiazine or the oral form of Terramycin were considered cured or improved. Terramycin intramuscularly appears to be of some benefit in the treatment of nonspecific urethritis, although inferior to sulfadiazine and Ter-

ramycin orally. Of those patients receiving no specific therapy, about 65 per cent were considered cured or improved; this points up the difficulty in evaluating any new type of therapy. Bacteriologic studies and the nature of the responses to the various antibiotics used lead one to conclude that the hemolytic staphylococcus may play an important role in causing this disease. Nonspecific urethritis is a disease that may have a rather prolonged course and that is subject to spontaneous remissions and relapses. That it is acquired by sexual intercourse is surmised but by no means proved. Because of the apparent frequency of some degree of prostatic involvement "prostato-urethritis" rather than "urethritis" would be a better term for the disease.

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When there is no explanation, they give it a name, which immediately explains everything.—Martin T. Fischer

PRINCIPLES OF SURGERY IN MANAGING MASS CASUALTIES

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ORDINARILY one writes an article with a certain amount of knowledge and experience as background, and there are recognized authorities whose pronouncements are relied upon. In the field of mass casualties, such as considered here, however, there are no experts and it must be borne clearly in mind that all who speak or write on this subject base their comments and judgments on analogy with civilian disasters or combat surgery. Such rationalizations may or may not be valid but are more apt to be acceptable than the naive and emotional comments made by academic rather than practical surgeons or by the stormy petrels who use this subject as another excuse to fly in the face of constituted authority. These words sound rather harsh, but in this new field where none of us can speak from experience it is too easy for false leaders to arise and becloud the issue. In a subject as complicated and potentially serious as that of surgery for mass casualties it is especially necessary to define our terms, speak accurately, avoid emotionalism, reassure the defeatists, silence the crackpots, stir up interest in the lethargic, do all in our power to arrive at a proper estimate of the probable situation, and make adequate, flexible plans on such a basis.

It must be emphasized again and again that the basic principles of management for cases of traumatic injury have been proved over and over. These principles may be modified somewhat to meet existing circumstances but will not change radically. All of our effort must be based on an accurate appraisal of the situation and current inventory of the personnel and materiel available, followed by proper triage of the casualties. What we are able to accomplish will be directly related to the number of casualties requiring care as compared to our capabilities from the standpoint of trained people and supplies.

It has been said that thermonuclear warfare is too horrible to contemplate and that, like the chemical agents, no one will dare use them for fear of massive retaliation. Possibly this is so, but no one has advocated that we neglect training to meet the threat of chemical warfare. Neither can we write off thermonuclear war-

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fare this casually. All of our effort in training may be like insurance against fire which does not take place or theft which does not occur. Nonetheless, mature men recognize the need for such insurance. Consequently, we must recognize and properly evaluate the problem, plan for suitable triage which is the core of treatment, set up plans for smoothly functioning organization, arrange for a workable chain of command with clearly delineated authority and responsibility, and finally we must implement a broad training program to cover all estimated needs.

RECOGNITION OF THE PROBLEM

It might be assumed that because much has been said and written about mass casualties, the problem is well recognized and delineated. This, however, is far from the truth, and there are three distinct schools of thought or lack of thought. First is the muddle headed school characterized by the oft-repeated statement, "We can handle the problem when it arises because we are used to treating cases of trauma every day." This statement, with its tragic implications, is heard often by those who are trying to get something accomplished in the way of civil defense.

For many years the American College of Surgeons has recognized that trauma cases, in general, are poorly treated; and it has been a common saying that it is safer to be wounded in battle than on the streets of one of our large cities. The Committee on Fractures of the American College of Surgeons was revised to become the Committee on Trauma in recognition of the fact that increased emphasis was needed on treatment of trauma in general. If medical practitioners handle individual cases of trauma poorly, what will they do when called on under adverse conditions to handle 50 cases, 100 cases, 1,000 cases, or 10,000 cases? Even physicians with previous military experience function poorly in a civilian disaster of such relatively small proportions as that at Worcester, Mass., in June of 1953, when there were 1,500 injured. The resulting fiasco indicates clearly what can happen when there is no prior planning, no co-ordinated direction of effort, and poor adherence to well-known surgical principles. For example, there was a frenzied interest in drawing blood, so that 950 units in excess of that which was needed were drawn, and because of lack of storage space, questions as to its sterility, et cetera, most of it had to be converted into gamma globulin later. Open fractures were primarily reduced and closed, penetrating head injuries were treated conservatively, patients were operated on without regard for shock, and surgeons worked far into the night by the light of flashlights when power failed, closing wounds primarily without debridement. The Worcester tornado has been well studied and officially documented and those who maintain that civilian physicians can take a disaster in stride are ignorant of fact.

Second is the academic school characterized by the belief that in a disaster all burn patients will have blood potassium determinations, individually calculated diets, massive occlusive dressings, and routine injections of various antibiotics, just as we do for individual patients in a teaching center. Such dreamers envisage evacuation of 100,000 casualties in 24 hours by helicopter to large hospitals miraculously empty of patients and with staffs and supplies ready and waiting. It hardly seems necessary to point out that most hospitals are always full to the doors, that usually they are located in an urban area which will be well within the zone of destruction, and that our available helicopters will accommodate two litters and one sitting patient. For our 100,000 casualties, therefore, allowing 30 minutes per trip and realizing that only daytime flights are possible, it would require 1,388 helicopters; or if we spread completion of evacuation over a three-day period, it would require 463 helicopters. There is not the remotest possibility that even a fourth of this number of helicopters would be available.

Third is the naive school characterized by the belief that we need not stockpile penicillin because we can buy it at any drugstore; or that all we have to do to obtain suture material is to go to the dime store for cotton thread; or that doctors, nurses, and supplies will materialize from surrounding villages in case of disaster to the city; or that there won't be any casualties anyway because we will all jump in our cars and head for the open spaces. In a recent small disaster there were about 30 casualties. The local community in a rather small town was completely incapable, and even with supplies and teams from a fairly distant large city, it was 72 hours before the last patient was operated on. In the recent Los Angeles train wreck there were about 150 casualties. Try to envisage the results of 100 such train wrecks simultaneously in the same city.

None of these seemingly fanciful ideas are fictional, and all have been advanced seriously by various individuals and groups. It would seem, therefore, that our most pressing problem is the injection of realism and common sense into our discussions, with emphasis on doing the best we can with the means at hand.

First, let us emphasize the sheer magnitude of the caseload. When Hiroshima, with a population of about 300,000, was devastated by our smallest A-bomb, 70,000 persons died, 15,000 were reported missing, and from 80,000 to 100,000 persons sustained injuries. This, of course, was in an untrained, unwarned population; but we have an untrained population, and who can say that we will have warning? Furthermore, newer weapons may do a hundred times the damage so that maybe the proportion of from 1 to 3 injuries per 10 population may not be out of line. For San Antonio this would give a figure of about 50,000 to 150,000 casu-

alties. San Antonio has only 40 per cent of its needed hospital beds now, and these always are filled. Even if all existing hospitals in San Antonio were miraculously spared, there would be no appreciable number of hospital beds available. We handle a slowly developing disaster like a flood fairly well, but there are few casualties in a flood, by comparison, and the analogy does not hold. Suppose we look at the personnel aspect of the case-load. On the average, one operating team will take care of 15 casualties in 24 hours, but let's step this up to 1 an hour for the sake of argument. This means that for our 50,000 casualties over 2,000 surgeons are needed, to say nothing of anesthetists, nurses, and helpers; or if we spread this out over 48 hours, we still need 1,000 surgeons. A minimal list of supplies needed to care for 1,000 mixed casualties for a 72-hour period has been drawn up and fairly well agreed upon. For travel over paved roads, this amount of supplies can be loaded into two 2½-ton trucks; for unpaved roads, 4 such trucks are needed; and if travel is to be over open terrain, 6 trucks are required. For our 50,000 casualties, therefore, 100 to 300 truckloads of supplies would be required. This means lots of loading, unloading, and gasoline consumption. At least 25 per cent of the casualties will have burns involving from 20 to 30 per cent of the body surface; and for 1,000 casualties with 30 per cent of the body surface burned, who are treated by occlusive dressings and with optimal fluids furnished, 5,712 pounds of dressings occupying 890.4 cubic feet plus 42,066.5 pounds of fluids occupying 15,281.3 cubic feet are needed. Figures for burns involving 20 per cent of the body surface are not significantly smaller. This means that for dressings and fluids alone, 1,000 burn casualties require seven 40-foot boxcars, and to treat our 12,200 burn casualties, therefore, 85 boxcars would be required. This is a train approximately a mile long. Can we presume that trackage will be available right up to the aid station or that the patients will conveniently lie down near the tracks? How many laborers and trucks will the unloading and transporting of these supplies require—presuming tracks and roads and laborers and trucks are available?

Suppose we are warned and that all of us jump into our cars to evacuate the city. This requires cars and gasoline, but also it presupposes a destination with food and shelter at the other end of the trip. Consider the situation if we add panic to the usual football-game traffic problem, or permit a hysterical mother to go across town to pick up the children at school. It has been said that if the people of San Francisco jumped into their cars and headed across the Golden Gate Bridge, 72 hours later the last car would get across the bridge and the head of the column would be entering Salt Lake City, Utah.

The problem is not one of everyday magnitude to be taken in stride as a routine day's work; nor will we be engaged in business as usual in our hospital centers; nor will we be giving maximum care to all casualties. This brings us to the matter of sorting patients, or triage.

TRIAGE

Proper sorting of patients is the essence of military surgery, and its aim is to categorize patients into groups such as those requiring immediate operation, those requiring treatment for shock or other resuscitation before operation, those who present no surgical emergency but can either be evacuated to the rear for care or can be kept a short time and returned to duty, and finally those whose condition is so serious that operation is not indicated. This last point brings up an interesting theological point. It has been said that in mass casualty work no doctor can "play God" and decide who is to die without treatment. Such a statement can never be made by a surgeon with any experience, either civilian or military, because a practical surgeon realizes that he accepts the responsibility for life and death as an unconscious part of his daily work. Every time the surgeon decides that a cancer patient has too much evidence of distant spread even to warrant exploration, he condemns that patient to death. At the operating table, when the surgeon decides that the lesion cannot be removed he likewise and just as definitely seals that patient's doom. Such decisions are legion and are not limited to the surgeon. Anyone with the slightest experience in combat surgery knows that certain patients who are obviously fatally injured are not operated on and this time is saved for a patient who can be benefited. This is not "playing God" but is a part of the day's work and is accepted as such by anyone capable of assuming the responsibility of being a surgeon. Let us not become emotional about this matter and let us not assume that it is something new associated only with mass casualty work.

Sentimental and inexperienced persons also raise ethical objections to triage of patients and pose hypothetical problems which may arise in the sorting of mass casualties. For example, the emotional cry of "save the women and children first" may be raised. Some wish to raise the economic and social status of the patient as a criterion. Here the battle cry would be "save the bank president first and let the janitor die." This could lead to absurd lengths and those who bring up such a matter obviously have had no experience and have not thought the problem through. Suppose there are 250 patients in severe shock and there is one-half pint of available blood, a not inconceivable situation. Shall we give each patient 1 ml of blood, or shall we eliminate from

consideration those patients who are beyond our help and concentrate our efforts on saving patients who are salvageable? In World War II prison camps, just such decisions had to be made, as for example when there were 100 prisoners with malaria and 100 tablets of quinine were issued. Do you give each man one tablet, which would do no one any good, or do you concentrate on a few of the men for better end results? It would not seem necessary to bring up such elementary points except for the fact that someone has to say these things in black and white for the sentimentalists and the inexperienced.

Obviously, we must stick to the basic principles of triage, selecting those patients whose condition warrants immediate operation, choosing those patients whose condition indicates resuscitation before operation, and segregating those patients for whom operation is not indicated because of the fatal nature of the condition. The heavy responsibility resting on such decisions indicates clearly why we have always said that the senior man in point of experience is the man for assignment as triage officer. Obviously, also, in mass casualty work, the triage officer must balance his decisions against availability of trained personnel and supplies. It will do no good to select patients for treatment of shock if there is no intravenous solution, nor will it be sensible to select patients for immediate operation if there are no surgeons. It seems logical to me that our treatment, in the event mass casualties are sustained, will of necessity be on a sliding scale depending on caseload and capabilities. What we need to do is to set up a system of priorities, listing the order in which case groups or procedures will be dropped from consideration as the caseload exceeds therapeutic capabilities. Here is the place for hardheaded practicality and a return to essential elements of basic principles. There is no place here for the sentimental slogan that every man must have the maximum in care and no casualty may be denied treatment. As a matter of fact, we may have to become considerably tougher in our thinking. What of the possibility of using cadaver blood for transfusion? Have any plans been made to include this? Is it possible that we might someday consider using the moribund and hopelessly injured as exsanguination blood donors? Such ideas are repugnant to us now but so is cannibalism until you are starving.

It seems unwise to attempt to set up such a list of priorities herein but certain general points can be stressed. For example, the sorting of patients is a constantly changing thing from the standpoint that the patient's condition changes and availabilities change. A patient in the resuscitation group may change to the immediate operation group when shock is overcome or may change to the "no treatment" group if shock is irreversible. Also, it may be that, faced with a tremendous caseload of abdominal injuries,

we would have to decide that we had neither the time nor the personnel to carry out abdominal exploration, and we would revert to conservative management. However, if suddenly a group of surgical teams arrive for duty, the triage is immediately revised and these patients enter the "immediate treatment" group, if their condition has not deteriorated beyond that point. The sliding scale and constant change with shifting circumstances makes it impossible to set out dogmatic lists and again places a premium on surgical experience and judgment.

Furthermore, presuming that an evacuation system will be possible, triage is repeated at each installation, again because of possible changes in the patients' condition or changes in personnel and supply availability at the different installations. It is re-emphasized that triage is not a final and one-shot decision but is synonymous in some respects with surgical judgment.

CHAIN OF COMMAND

The moment a triage officer begins to select patients in a civilian disaster for immediate operation or for resuscitation, the concept of chain of command comes up, because every civilian physician within miles will at once question his authority and ask by what right he directs therapy. One of the most difficult points to put across to civilian physicians is the need for teamwork and division of labor for the common good. Each one demands for himself the right to decide what therapeutic procedures he will use and totally rejects what he considers meddling in his business, whether or not he is qualified, being irrelevant to him. We in the military service know the necessity for teamwork and the necessity for at least minimal standardization of therapeutic procedures. In the service it is simple to arrange that only the qualified receive assignments requiring special skills, and probably our patients would do even better were we to exercise more supervision over methods of treatment. In time of disaster, it is essential that previously designated leaders assume control as early as possible to prevent waste of supplies and professional effort. What can happen in the absence of qualified leadership is observed at the site of an automobile accident where a crowd immediately forms. Some want to have the victim lie down with feet elevated, some want to support him and have him walk around, some want to loosen his clothing, others want to apply a splint. The Worcester tornado again is a good example of what should not happen. Without leadership, one hospital became completely overrun with patients and relatives while another hospital nearby received almost no admissions. Doctors climbed over patients in the ambulances, trying to treat them instead of establishing a flow of litters to the emergency room. There was no designated authority and, therefore, no co-ordination between the blood collectors and the blood users.

At present, there is no one to assume command in civilian communities should a disaster strike, and usually the armed services are called in to organize the effort. It seems possible that in the event of mass casualties, martial law will be declared, thus making the military responsible. Therefore, it is highly desirable that we be ready and prepared to assume this role. Unfortunately, there is no current mechanism whereby any person or group can exert any pressure to get civil defense rolling. If a city does not have the urge to prepare, it simply ignores the problem. Anyone who has ever tried to speak on civil defense to a civilian audience realizes the depth of the apathy which exists. All of our wars in recent years have been fought on foreign shores, why not the next one? None of our cities have ever been bombed, why worry about such a remote contingency? If anything is to be our undoing, it will be the disinclination to appoint responsible leaders and follow their directions.

ORGANIZATION

It is all very well for surgeons to talk about what they will do in the event of mass casualties, but actually nothing will be accomplished unless there is a very efficient, preplanned organization which will get the doctor to the patient or the patient to the doctor, assure a steady flow of supplies, provide for evacuation of patients, and do the hundred and one things which the surgeon will be too busy to bother with. This preplanned organization is familiar to the military men but is unfamiliar and repugnant to civilians who proudly rebel at "regimentation" or interference with their "freedom of action." Without organization, in the event of disaster, they will be free to die and that is about all. The plans must include provision for warning and continuing communication so that orders and directives may be disseminated. Block and area wardens must be ready and previously trained to supervise rescue work and first aid as well as police protection and fire fighting. Very early, engineers must be ready to bulldoze roadways through the rubble, and then immediate traffic control will be essential. According to a preconceived plan, litter teams and other conveyances then can start evacuating patients to aid stations where triage, resuscitation, and first aid will be done and where food and shelter will be provided. Here, or close by, the medical regulating officer must direct evacuation to designated emergency hospital areas or special treatment centers. During all this, the supplies must be coming forward, immunization and public health matters must be taken care of, and welfare work must be progressing. This is indeed a complicated organizational setup but one which is routine for the combat forces, and the civilian group might well copy the military organization in the event of mass casualties. Such an organization does not spring up overnight, nor does it develop like Topsy without direction.

TRAINING PROGRAM

The training program must be an extensive one, covering all of the functions listed in the previous section on organization, and most persons in the civilian community could well afford to become proficient in at least one of these fields. In addition, every individual should be qualified in first aid and the elementary technics of survival. It is true that the American Red Cross and the Boy and Girl Scouts have shown interest in first aid, but this should be pushed vigorously so that every individual is qualified. First aid and self-help may be lifesaving in an emergency.

Some plans have included formation of definitive teams such as surgical, shock, orthopedic, et cetera, similar to the teams of the Auxiliary Surgical Group of World War II. Currently, we consider that such teams are wasteful of personnel and most are unnecessary. Furthermore, in the holocaust of disaster the team members may be widely separated and it would be better to have the members basically qualified in first aid, insertion of an intravenous needle, giving of a hypodermic injection, sterile technic at the operating table, functions of a circulator, and possibly administration of ether under supervision. Formation of rigid teams with clearly delineated jobs may be too specialized for the needs.

It seems necessary to mention specifically some things in which laymen should not be trained. Currently there is some thought that large groups of people should be taught how to do a tracheotomy. This seems to me to be entirely unsound and dangerous. We find that very few physicians have actually performed tracheotomy and relatively few are sure of the indications. How, then, can we expect laymen to know when to do the operation even if they know the technic? This seems to me to be dangerous and an evidence of muddled thinking. We have always had the same difficulty with such an apparently simple thing as the tourniquet. You can teach any moron *how* to use a knife but can you teach him *when* to use it? We would do well to teach things to laymen which do not depend on professional judgment.

Finally, it seems likely that in the event of mass casualties there will be a shortage of surgeons, and we envision one surgeon acting as triage officer while another supervises as many as six operating tables where physicians who are not trained surgeons are doing such things as wound débridement. This supervisor could actually take a hand in more technical procedures, still being free to oversee the other tables. In this way, while professional competence might be somewhat lacking, at least a greater volume of work could be turned out than by one surgeon alone.

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SUMMARY AND CONCLUSIONS

Because there are no experts with experience in managing mass casualties, many impractical and academic thoughts have been expounded as gospel.

The basic principles of military surgery, based on proper selection of patients, still are sound.

Mass casualties still will be composed of the types of injuries we are accustomed to treat, and the difference will be in the extremely large numbers and the suddenness of their occurrence.

Proper sorting of patients and decision as to what can be accomplished must be based on an accurate estimation of the situation from the standpoint of the caseload requiring care as contrasted with the availability of trained personnel and supplies.

Adequate functioning in time of disaster will depend on prior planning, a definite chain of command with delineation of authority, and a suitable organization which takes into account all of the elements needed to find, evacuate, resuscitate, and treat the injured.

A training program, broad in scope but not requiring surgical judgment of laymen, is essential.

Less talk, more common sense, and some action would seem to be highly desirable.

THE ENERGY OF THE A-BOMB

The energy of a nominal A-bomb (equivalent to 20,000 tons of TNT) is 2×10^{13} cal. This is divided into thermal and kinetic energy although the latter will also ultimately be converted into thermal energy. The thermal energy from the sun falling on 1 square mile of Nevada ground during an average spring day supplies as much heat as two nominal bombs. The energy released by the condensation of water in a typical thunderstorm is equivalent to 13 nominal bombs. Further comparisons with natural phenomena reveal similar statistics suggesting that the energy of an A-bomb, while tremendous compared with the energy of other man-made explosions, is relatively small compared with that of many natural phenomena.

—L. MACHTA and D. L. HARRIS
in *Science*, p. 78, Jan. 21, 1955

Clinicopathologic Conference

William Beaumont Army Hospital, Fort Bliss, Tex.*

ABDOMINAL PAIN AND ANOREXIA

Summary of Clinical History. A 25-year-old male veteran was admitted directly to this hospital on 17 September 1953 from a civilian hospital because of a three-week history of weight loss and pain in the abdomen and back.

He had worked as a filling station attendant since discharge from the Army in April 1953. His past health had always been good. Family history was noncontributory. About 1 September 1953, he developed a constant severe bilateral low lumbar backache. In a few days this pain seemed to radiate around both flanks to the abdomen. There was no relation to food intake and no nausea, vomiting, or bowel dysfunction. His appetite fell to nothing. He was admitted to a civilian hospital on 3 September 1953 and treated initially with salicylates for a strained back. It was necessary to use narcotics to control abdominal pain. At this hospital roentgenograms of the chest, abdomen, and spine revealed no abnormalities. A barium enema and gastro-intestinal roentgen series were likewise negative. Intermittent fever and constant polymorphonuclear leukocytosis were noted. The patient lost about 20 pounds in weight, going from 150 to 130 pounds in the two weeks prior to admission to this hospital.

Physical Examination. The patient was a poorly nourished young man in marked distress from back and abdominal pain. The height was 6 feet; weight, 130 pounds; temperature, 101°F; pulse, 110

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per minute; and blood pressure, 120/70 mm Hg. The tongue was dry and oral hygiene poor. Examination of the head and neck was otherwise not remarkable. The optic fundi appeared normal. The lung fields were clear to percussion and auscultation, and the heart was not enlarged to percussion. The sounds and rhythm were normal except for tachycardia. No adventitious sounds were heard. The abdomen was flat and generally tender. There was no localized tenderness or true rigidity. The liver, spleen, and kidneys could not be palpated and no masses were felt. Rectal and genital examinations were normal except for a small, soft nodule above the right testis. The back showed paravertebral and sacroiliac tenderness with limitation of all low-spinal movements due to pain. The extremities showed no abnormalities, and neurologic examination was within normal limits. Slightly enlarged lymph nodes were felt in both axillae but no other lymphadenopathy was noted.

Laboratory Studies. On admission urinalysis was normal. The specific gravity was 1.015; sugar and albumin negative, and only an occasional white blood cell on microscopic examination. The hemoglobin was 12.3 g/100 ml; the red blood cell count, 4,090,000/μl. The white blood cell count was 28,000/μl, with 92 per cent polymorphonuclear leukocytes, 5 per cent lymphocytes, 1 per cent monocytes, and 2 per cent eosinophils. The sedimentation rate was 36 mm/hour (corrected) by the Wintrobe method. The serologic test for syphilis was negative. The spinal fluid contained no cells, and chemical and serologic studies on the fluid were normal. Repeated stool examinations showed no blood, parasites, or ova. Blood cultures were sterile. The white blood cell count and sedimentation rate remained elevated. Urinalyses remained negative. Agglutinations for Brucella were negative, and those for typhoid H and paratyphoid A showed low titers of an immunized individual. Serum amylase was 64 units. Gastric analysis revealed 6 units of free hydrochloric acid. Urine examinations for porphyrin and porphobilinogen were negative. Liver function tests were normal except for a cephalin-cholesterol flocculation of 3 plus in 24 hours and a thymol turbidity of 12.7 units. Bone marrow aspirated from the iliac crest showed mild myeloid hyperplasia. Roentgenograms of the chest, abdomen, and spine were normal. Cholecystograms, intravenous urograms, a barium enema, and an upper gastro-intestinal series revealed no abnormalities. An electrocardiogram was normal.

Course in Hospital. During the first month of hospitalization the patient remained febrile, the temperature spiking from 99° to 102.5°F daily. He complained more and more of severe upper abdominal pain. This was intermittent, occurring three or four times daily, and was relieved only by narcotics. There was no sign of peritonitis or localized abdominal infection. Penicillin

and streptomycin sulfate were given empirically for several weeks. On 2 October 1953, a single white blood cell differential count showed 13 per cent eosinophils. On 4 October 1953, he complained of numbness and aching in the feet and the calf of the left leg. Marked weakness of dorsiflexion of the right foot was noted, and the Achilles jerk reflex was absent on the left. An area of hypesthesia appeared on the right ankle. These findings slowly cleared only to be followed by transient left ulnar palsy.

On 6 October, specimens for biopsy were taken from a lymph node from the left axilla and from the left pectoralis major muscle. Both specimens were normal, showing no pathologic changes on microscopic examination. A biopsy specimen from a right axillary node later showed only nonspecific hyperplasia.

By 18 October, the patient weighed 108 pounds. At the suggestion of a consultant, who felt that amebic abscess was not excluded, the patient was started on a course of emetine hydrochloride. He received 1 gram daily intramuscularly for six days. During this time fever subsided slightly. Electrocardiograms showed no abnormality. After the course of emetine he was given Terramycin (brand of oxytetracycline) intravenously for several days in hopes of treating any concealed infection. On 26 October 1953, three days after emetine was discontinued, he developed a gallop rhythm. Electrocardiogram showed flat T waves in the limb leads and inversion of T waves in precordial leads V_2 and V_6. A roentgenogram of the chest showed an increase in the heart size and congestive changes in the lung fields. A brief episode of pulmonary edema was precipitated by the intravenous administration of fluids. This responded promptly to morphine, oxygen, digoxin, and Mercuhydrin (brand of meralluride sodium). Electrocardiograms continued to show a pattern of myocarditis though digoxin was discontinued.

On 5 November 1953, he was transferred to the Surgical Service in preparation for exploratory laparotomy. His temperature had returned to normal, but leukocytosis was still found, and the severe abdominal pain continued. Nasogastric suction was applied with little relief of his pain. Transfusions of whole blood were given prior to operation because of mild anemia.

During the last part of the operation the patient became severely hypotensive and cyanotic. Abdominal closure was performed as quickly as possible and he was given 20 ml of aqueous adrenal cortex extract intravenously, which returned the blood pressure to normal. He returned to the ward in fair condition and started taking fluids orally by the third day. Electrocardiograms during the first postoperative week indicated that an anterior myocardial infarction had been sustained. Immediately after operation the

blood pressure began to rise, reaching an average level of 160/120 mm Hg. Along with this development albuminuria appeared for the first time.

DISCUSSION

Doctor Abildskov:* This is a difficult case because in spite of the length of the protocol there are very few positive findings. We have a great number of findings, but most are on the negative side of the ledger and are not very helpful in arriving at a diagnosis. We could name a good many things the patient probably does not have, but this is not a productive approach in the absence of enough positive findings to point toward the correct diagnosis. With the few positive findings which are available I am not even certain whether this patient's disease was neoplastic, degenerative, inflammatory, or toxic, and actually, diagnoses from all of these categories can be considered. To emphasize the sparsity of positive findings I would like to list all of those that I found in the protocol.

First of all, we know the patient was 25 years old. In general we tend to think of different disease entities in 25-year-old patients than we do, for example, in 65- to 75-year-old patients; but in this case even this type of reasoning does not appear to apply since the patient is said to have developed electrocardiographic evidence of myocardial infarction, and this is not one of the entities commonly considered in 25-year-old patients. Secondly, we know something about his occupation. Occupational history may be extremely significant and again may be completely unrelated to a patient's illness. For what it is worth, we know that this patient was a filling station attendant prior to the onset of his illness. We also know that his illness had a relatively sudden onset. His initial symptom was pain in the lumbar region and he later developed abdominal pain which became a prominent feature of his illness. The patient was anorexic and had a weight loss amounting to 20 pounds in the two weeks preceding admission to this hospital.

As far as physical findings are concerned, there were very few positive ones. His illness was characterized by fever with the temperature on occasion rising as high as 102.5°F. Apparently his abdominal tenderness was not well defined, and the vague nature of this tenderness may actually be of help in arriving at a diagnosis. A small nodule above the right testicle is described but is not mentioned again. We do not know what happened to this nodule during the course of his illness. He had sacro-iliac tenderness, pain on motion of the lumbar spine, and slightly enlarged axillary nodes. Later in the course of his illness he had a rising blood pressure, cardiac enlargement, gallop rhythm, and other evidences of congestive failure. One of the most interesting findings was a transient peripheral neuritis involving the ulnar nerve and producing a definite foot drop.

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From the laboratory we have what is probably the most striking feature of his illness, namely, a consistent and marked leukocytosis. The white blood cell count was as high as 28,000/μl with a marked increase in polymorphonuclear leukocytes, and, while it is not mentioned, I presume a marked shift to the left. He also had a mild anemia, but we are not given sufficient data to define the anemia morphologically and probably the morphology would not be especially helpful. He had an increased sedimentation rate, minimal evidence of liver dysfunction manifested by a cephalin-cholesterol flocculation of 3 plus and a thymol turbidity of 12.7 units. A bone marrow examination was done and I think this examination is extremely important in ruling out several diagnostic possibilities which might otherwise be considered in this case. The bone marrow showed only mild myeloid hyperplasia and probably cannot be related in any specific way to the illness. One white blood cell count only, out of many reported, showed a marked eosinophilia. Urine examinations were consistently negative during the initial phases of his illness, but he later developed albuminuria. Coinciding with the development of clinical evidence of cardiac disease, he developed electrocardiographic changes of a nonspecific type and later is said to have had the electrocardiographic findings of acute myocardial infarction.

These are all of the positive findings I was able to get out of the protocol. On the negative side we have a host of findings, but I will not attempt to evaluate the significance of each of these. Some of the more important negative findings include the absence of splenomegaly, hepatomegaly, and more marked and definite lymphadenopathy. The absence of these findings is helpful in leading us away from several specific illnesses but is not particularly helpful in leading us toward a definitive diagnosis.

Approaching this case from the standpoint of what must have appeared to be the major positive findings, namely, abdominal pain, tenderness, fever, and leukocytosis, there are a great many diagnostic possibilities. For example, acute appendicitis followed by rupture and abscess formation would explain many features of this illness. There is nothing, however, which leads us to particularly favor that diagnosis over several other possibilities. We should also consider acute cholecystitis. He did have, at some stages of his illness, right upper quadrant pain, and certainly this disease process commonly causes such pain, leukocytosis, and fever; again, however, nothing leads us to particularly favor that diagnosis. Pyelonephritis should probably be considered in a patient with back pain, leukocytosis, and fever; however, in the absence of urinary findings early in the course of his illness this becomes very unlikely.

If we consider some of the less prominent features of this patient's illness we are led to consider some of the less common disease processes. The patient had a nodule which is described as lying above the

right testicle. Is it possible that this represented a testicular neoplasm and could such an entity account for any or all of the features of this patient's illness? Neoplasm, wherever it arises, the testes being no exception, can certainly cause febrile illness. No objective evidences of metastasis were found; however, I think this type of disease should be listed among the diagnostic possibilities.

This patient had a debilitating disease with weight loss and fever, which fits the description of Pel-Ebstein fever. Hodgkin's disease certainly must be considered; however, in this case we have the information that a biopsy specimen of a lymph node did not show any pathologic changes. If Hodgkin's disease is present, it is probably localized in the abdomen. This diagnosis has the additional virtue of possibly explaining the peripheral neuritis. This is an infrequent finding but has been reported in patients with Hodgkin's disease.

In considering a patient with an obscure febrile illness, tuberculosis should be mentioned. Again, there is nothing which particularly favors this diagnosis over several others but it must be placed on the list of possibilities.

Some of the other unusual features of this patient's illness were a rising blood pressure, cardiac enlargement, gallop rhythm, transient peripheral neuritis, and the occurrence of albuminuria. All of these findings may be unrelated to the primary illness. For example, the elevated blood pressure and the albuminuria occurred following an episode of shock during an operation. These could be the result of acute renal failure or lower nephron nephrosis. The cardiac enlargement, gallop rhythm, other evidence of congestive failure, and the electrocardiographic findings occurred a few days after emetine therapy had been given. If these cardiac findings were the result of emetine toxicity, it is surprising that electrocardiographic changes did not occur during the course of therapy.

If all or most of the findings in this case are the result of the primary disease, one particular diagnostic possibility seems to explain them better than any other entity. This is not a diagnosis which is established clinically. It is a diagnosis which is to be suspected clinically, but confirmation depends on biopsy. This entity is periarteritis nodosa. This disease can certainly produce an illness with an acute onset. More commonly it has an insidious onset, but in a significant number of cases the onset is explosive. Patients who are eventually found to have periarteritis nodosa not infrequently are subjected to laparotomy, at which time the diagnosis is suspected from the appearance of the blood vessels or is confirmed by examination of a biopsy specimen of, or removal of, an organ such as the gall bladder. This diagnosis would account for the febrile nature of this illness and the severe abdominal pain and tenderness. Pain in the back is a fairly common finding due to periarteritis nodosa, as is a moderate degree of lymphadenopathy.

This is the single diagnosis which, it seems to me, will link the initial features of this patient's illness with the positive findings that developed during his course in the hospital. Hypertension, albuminuria, and the cardiac findings are all consistent with this diagnosis. Approximately half of the patients with periarteritis nodosa develop hypertension, and this finding is one of the excellent physical signs helping to substantiate this diagnosis. This diagnosis would also account for the otherwise puzzling finding of acute myocardial infarction in a 25-year-old man. Peripheral neuritis also fits very nicely with this diagnosis. Commonly, the patient with periarteritis nodosa will complain of pain in the legs; however, this patient did have definite peripheral neuritis involving both the lower and upper extremities.

Most of the laboratory findings given are consistent with the diagnosis of periarteritis nodosa. Leukocytosis of this degree is not the rule but on the other hand is not unheard of. Mild anemia is a fairly consistent finding in this disease, and minor abnormalities in liver function are well known to occur during its course. This diagnosis could also account for the eosinophilia, which is often transient, and for the albuminuria, which is a fairly consistent finding at some time during the course of periarteritis.

I think we should also consider some other diagnostic possibilities although none of these appear to me to account for the findings in this case as completely as does periarteritis nodosa. For example, the diagnosis of amebiasis and amebic liver abscess should be considered. This diagnosis was actually strongly considered during life, and the patient was given a therapeutic trial of emetine. Since we now know that there was no dramatic response to emetine, this diagnosis does not appear to be as likely as it must have appeared during the course of the patient's illness. Pyogenic abscess of the liver must also be considered, as must other localized accumulations of pus in the abdominal cavity, but again there is nothing which leads us particularly toward those diagnoses. One other possibility which should be strongly considered relates to this man's occupation. We know that he worked in a filling station, that he had an illness in which fever and abdominal pain had prominent features. Under these circumstances lead poisoning certainly appears to be a possibility.

Doctor Martin:* At operation the patient was found to have some obviously abnormal vessels in the region of the gall bladder; these were sclerotic and almost white. The gall bladder was removed, mainly because of these abnormal arteries, and not so much because it looked like a "hot" gall bladder. The early postoperative period was marked by the development of hypertension and the appearance of albuminuria. There was not much relief from the abdominal pain that was to dominate

*Maj. Thomas S. Martin, MC, USA, Assistant Chief, Department of Medicine.

the picture for the rest of his days, a span of about seven months. During that time he did not have a steadily progressive course but had some ups and downs which may well have been related to therapeutic effort.

About two weeks after operation 100 mg of cortisone a day was begun, which caused very definite reduction of his toxic symptoms, fever, anorexia, leukocytosis, and eosinophilia. The red and white blood cell counts returned to normal, and during December he gained nearly 20 pounds, reaching approximately 103 pounds. There was some lessening of abdominal pain. However, as might have been expected, there was no improvement in the albuminuria or hypertension. Late in December he "escaped" from the cortisone therapy, whereupon pain and anorexia increased and he again began to lose weight. The blood pressure was now running in the region of 190/130 mm Hg, and early papilledema had appeared, with retinal exudate. His urine was consistently showing 3 to 4 plus albuminuria and casts.

He was then begun on hexamethonium and Apresoline (brand of hydralazine hydrochloride) orally. Mercurials were used successfully with low salt intake to combat mild congestive failure that had taken place. On the antihypertensive agents there was a return of the blood pressure to levels at the upper limit of normal. With this there was considerable improvement in appetite and tendency to gain weight returned. The opiate requirement fell from four or five injections a day down to one or two. Then in early February there occurred a pneumonitis with chest pain and consolidation in the right lower lobe and fever. Bacteriologic and bronchoscopic studies were negative, and it was not certain whether the involvement was infectious or arteritic. But he was taken off cortisone and placed on penicillin and streptomycin. The lung involvement apparently disappeared, but the rest of the picture became worse.

In early March cortisone was re-instituted at the level of 75 mg a day, and there was some general improvement. However, the hypertension became worse. The parenteral form of hexamethonium was then begun in place of the oral form and the dose increased to a maximum of 50 mg every eight hours. This proved effective and the azotemia which had developed disappeared, the retinal lesions regressed, and the tendency to pulmonary edema subsided. However, this time, although the cardiovascular signs and symptoms improved, abdominal pain and anorexia remained unchanged.

His physician decided that a good deal of the complaint might have been due to opiate addiction and with considerable difficulty weaned the patient off all opiates except one or two doses of codeine per day. This was followed by the best three weeks (the first three weeks of April) that the patient had had since entering the hospital. His weight returned to a little over 100 pounds, albuminuria all but disappeared, papilledema disappeared, and anemia regressed. He was up and about

for several hours a day. Then in later April the flank and abdominal pain became worse, and he was soon taking three to four shots of morphine or Demerol (brand of meperidine hydrochloride) daily. His nutritional state rapidly worsened again.

All sorts of measures were tried to find the organ or organs which were the seat of the pain and to relieve it. It was presumed the pain was caused by ischemia due to impaired arterial blood supply, but the patient never showed any gross signs of infarction of the bowel such as bloody stools or change in bowel habits. Liver function tests remained reasonably normal. There were no changes in sensation over the abdomen to indicate neuritis in the nerve supply of the abdominal wall. He did develop a sudden, severe pain in one testicle that subsequently atrophied; this was considered to be evidence of occlusion of an artery on that side. He was tried on a period of parenteral nourishment only. A trial of 300 mg of cortisone per day for several days instead of the usual 75 mg accomplished nothing. Hexamethonium was removed temporarily, but this measure also was unavailing and only allowed a prompt rise in his systolic and diastolic pressures. Restoration of hexamethonium was again effective in controlling the blood pressure. However, during May he apparently became insensitive to hexamethonium and, even though its administration was pushed to the point of bowel and bladder stoppage and Urecholine (brand of bethanechol chloride) was used, no drop in blood pressure could be noted following an injection. The drug was therefore discontinued.

The great pain in the abdomen and flanks and a substernal burning with dysphasia (which was not relieved by antacids or massive vitamin therapy) dominated the picture until the final phase of his illness. On 29 May the anesthesia department, in response to a plea for help in alleviating the severe pain, performed a subarachnoid alcohol block of the lower dorsal and upper lumbar segment. During the next two days the patient was satisfied with only one injection of Pantopon per day. Then on the night of 31 May he seemed confused and was careless of his privacy, urinating on the floor. The next morning he was found to be stuporous and shortly exhibited a left-sided Jacksonian convulvion. The blood pressure was 204/150 mm Hg, and papilledema was evident. Hexamethonium was resumed and was found to be effective, and it was simple to keep his blood pressure at a reasonable level.

For the remaining six days of his life he never regained consciousness, although he showed a slight evolution toward a conscious state, so that before his death he had begun to respond to slight stimuli, look around, clear his throat, cough, et cetera. After the institution of anticonvulsive medication the first morning that he exhibited a seizure, convulsions did not recur. However, he continued to show twitching of the face and, to some extent, of the arms. It occurred to us that the twitching and perhaps some of the other neurologic findings might be due in part to opiate withdrawal. However, a large dose of Demerol did not affect any of the signs. We thought he had had some small hemor-

rhages in the right motor cortex and internal capsular area. He died early on the morning of 7 June without a preceding downward trend in the vital functions, suggesting that he had had a fresh cerebral vascular accident.

Dr. Abildskov's diagnosis:
Periarteritis nodosa

PATHOLOGIC FINDINGS

Doctor Heslington:* The operation performed was a cholecystectomy. The gall bladder was of normal size but with a slightly thickened wall. The mucosa was ulcerated inferiorly and the ulcer was covered by adhering brown crystals. The cystic artery and its branches were greatly thickened with pinpoint lumina (figs. 1 and 2). No aneurysmal dilatation was seen. The most striking microscopic change was in the vessels,

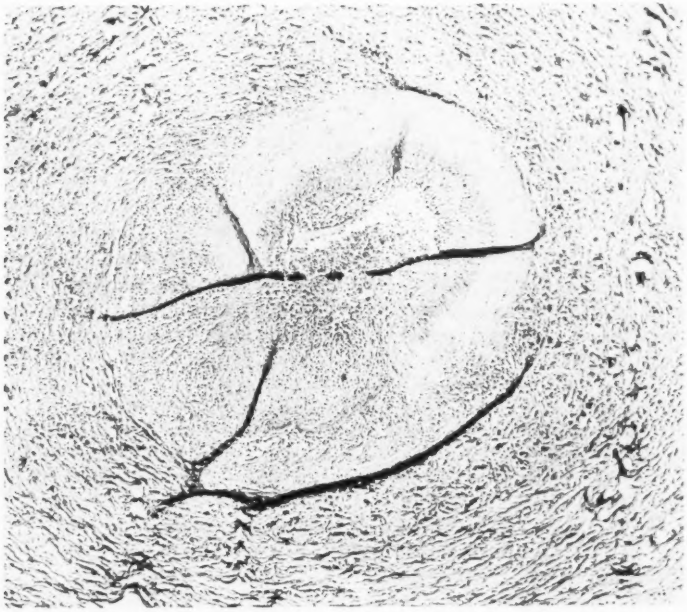

Figure 1. Section of cystic artery, showing necrosis of a portion of the wall with exudate in and about the wall. Adventitial thickening is extreme.

mainly the small elastic and muscular arteries. These showed spotty but widespread periarteritis nodosa. The lumina of the vessels showed marked to extreme narrowing, and some were thrombosed and recanal-

*Maj. Hurston F. Heslington, MC, USA, Chief of Anatomic Pathology Section, Laboratory Service.

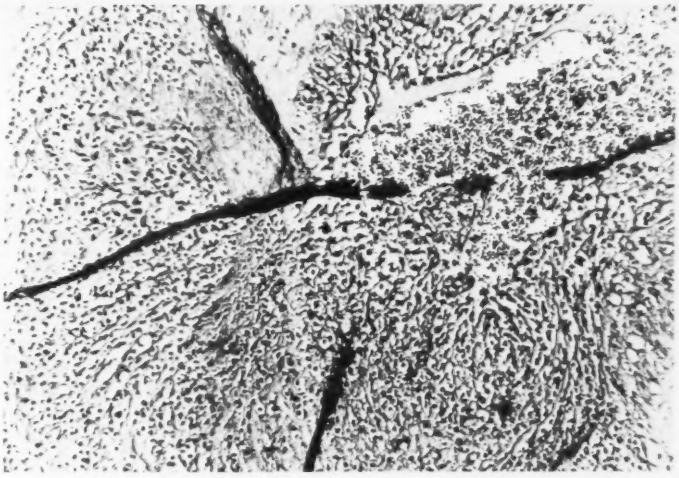

Figure 2. Higher power of section shown in figure 1 showing abundant fibrin and numerous histiocytes and polynuclears with necrosis.

ized. Active lesions were also present. In these there was necrosis of a part of the wall, usually including the muscularis, with partial replacement by fibrous tissue and inflammatory exudate. Histiocytes predominated in the exudate, but polynuclear leukocytes were numerous. The adventitia of the vessels was extremely thickened and infiltrated with inflammatory cells similar to those seen in the wall proper. The lesion at times involved almost the entire circumference of the vessel, particularly the adventitial thickening. In other instances the changes were eccentric, involving only a portion of the wall. Similar but less marked changes were present in some veins. The lesions varied from acute to healed. The mucosa of the gall bladder was somewhat thickened, and there was increased pigment in the epithelial cells. There were heavy deposits of pigment overlying the mucosal ulcers. These were apparently secondary to ischemia as the result of arteritis. The diagnosis was essential polyangiitis of the cystic arteries and veins.

At autopsy the periarteritis was seen to involve the vessels of the heart, lungs, pancreas, adrenals, kidneys (fig. 3), and brain. In all these organs there was evidence of infarction or softening. The characteristics of the vessel lesions were a patchy distribution with segmental involvement, involvement of large muscular arteries, particularly about their points of branching, and marked variation in the ages of the lesions. Those in the gall bladder wall were acute with marked exudative features, while at autopsy most of the lesions were quiescent or active with little evidence of exudation. The lesions in the kidney showed a

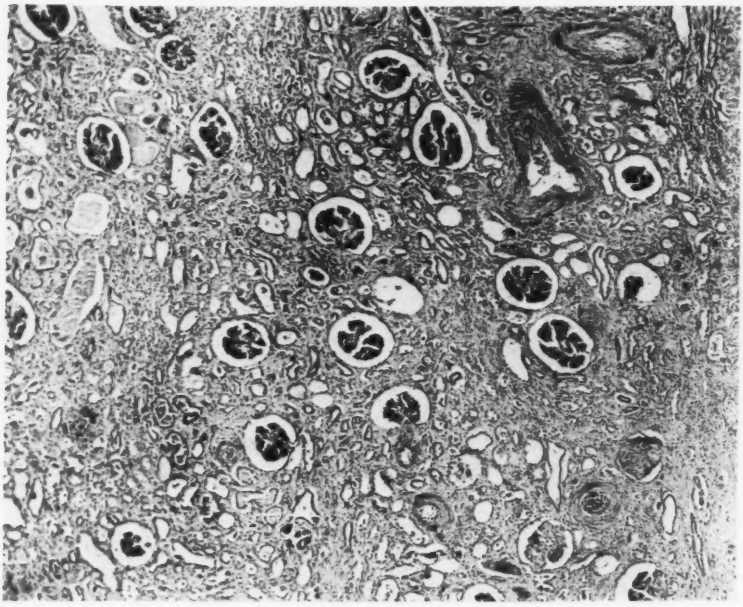

Figure 3. Low-power photomicrograph of section of kidney, showing vessel lesions, infarction, interstitial fibrosis, atrophy, and dilated Bowman's spaces.

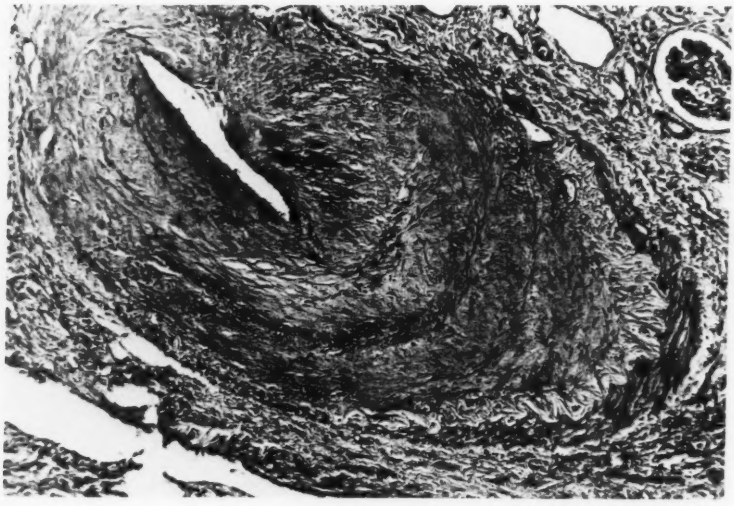

Figure 4. Section of arcuate artery of kidney, showing lamellar thickening of intima and partial to complete replacement of the muscle. Some necrosis, but exudative features minimal.

replacement of the muscle by fibrous tissue with little necrosis (figs. 4 and 5). The pictures of the vessels in the gall bladder and of those in

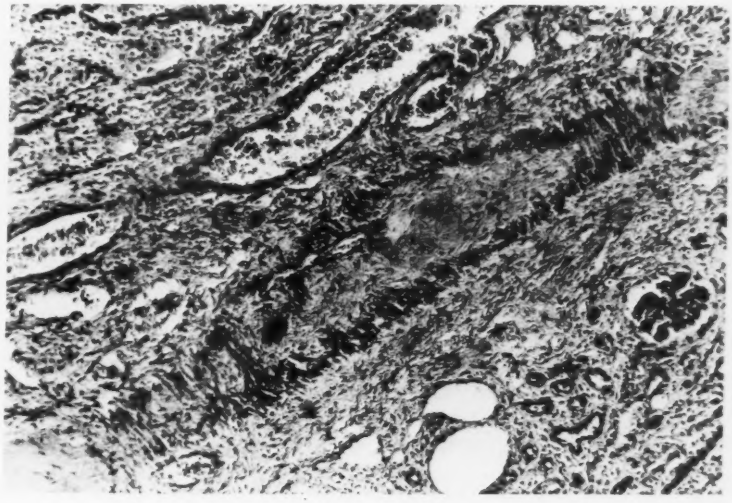

Figure 5. Longitudinally cut arteriae rectae in kidney, showing changes similar to those in the arcuate artery.

the kidney illustrate well the differences in the two lesions. Superficially, the lesions of the renal vessels resemble somewhat those seen in thromboangiitis obliterans. It is of some interest that the vessels of the gastro-intestinal tract showed no involvement, either grossly or microscopically.

Pathologic diagnosis:
Periarteritis nodosa

It takes fifty years from the discovery of a principle in medicine to its adoption in practice.—Martin T. Fischer

SERVICE ARTICLES

EMOTIONAL STATUS OF THE U. S. SOLDIER AND LENGTH OF TOUR IN KOREA

ARTHUR ROBBINS, *First Lieutenant, MSC, USA*

A CURRENT problem is determination of the deleterious effect of the length of tour in Korea on the mental health of the enlisted man. Complaints of boredom by enlisted men and officers alike have often been made to both staff and command officers concerning the 16-month tour. The separation from family and loved ones, the over-all restriction, and the long, arduous routine of field duty have all been pointed out by medical personnel as contributing to a pervasive strain on the personality structure. The goal of this study is to shed an objective light on the relationship, if any, between a number of emotional indexes as found in psychologic examinations and duration of stay in Korea. More specifically, we wish to explore the relationship of thematic apperception themas and psychosomatic check-list complaints to the months of duty percentwise completed in this area by the United States soldier.

Two hundred U. S. soldiers were selected at random from the entire 35th Infantry Regiment. At the time of examination this particular organization was in reserve. All subjects expected to spend at least 14 months in Korea. In order to have a relatively equal distribution for comparison, the total population was first broken down into quartiles (according to length of tour completed), and then 50 were selected at random from each group.

Two principal instruments were used in the study. A psychosomatic check list was extracted from the Hypochondrias Scale of the Minnesota Multiphasic Personality Inventory. Interspersed with these items were a number of buffer questions to make the list as unstructured as possible. A Thematic Apperception Test was also administered to evaluate some of the needs and presses operating within the soldier. The instrument consists of a number of pictures about each of which the subject is requested to make up a story. The principle underlying the instrument is that, in relating a story, a person organizes material from his own experiences, immediate perception of the stimuli, and the associations to those perceptions selected from conscious imagery. Cards 1,

From 544th General Dispensary, APO 503, San Francisco, Calif. Dr. Robbins is now at Family Consultation Service of Eastchester, Tuckahoe, N. Y.

2, 3, 4, 6, 7, 8, 12, 13, and 18 were used for this purpose. The following variables were analyzed by the examiner:

1. Physical aggression
    a. Nondirected
    b. Against the male
    c. Against the female
    d. Against the male and female
2. Verbal aggression
    a. Nondirected
    b. Against the male
    c. Against the female
    d. Against the male and female
3. Affiliation
4. Abasement
5. Escape
6. Sexual misidentification
7. Lack of past
8. Lack of outcome
9. Constriction

Finally, an information form requesting various identifying data was filled out by each enlisted man. Table 1 gives the various descriptive data of 191 U. S. soldiers. Nine subjects were lost from the original 200 because of rest and recuperation leave, temporary duty, or other special assignments. Unfortunately, there were little, if any, comparative statistics available to judge the normality of our sample. One may note, however, that the group appears to be a fairly typical one. Thus, the greater part of the sample fell in the age group of 18 to 21, were largely privates, of the Caucasian race, and were, in the large part, Protestants. In addition, the average soldier of this sample was single, of average intelligence, of near high school education, and lacking in combat experience.

PROCEDURE

During the latter part of May 1954, a research team* composed of one clinical psychology officer, four clinical psychology technicians, and one social worker technician visited the 35th Infantry Regiment. The subjects were informed that the study was part of

*1st Lt. Arthur Robbins, MSC, USA, Project Coordinator; 1st Lt. Leonard Pearson, Assistant Coordinator; Pvt. Wilmer R. Cozens, Chief Statistician, PFC Jack Kilberg, Assistant Statistician, Examiner; Pvt. James Hearst, Judge, Examiner; PFC Robert Elliot, Outside Judge, Examiner; PFC Joseph Kennedy, Examiner, Assistant Statistician; and Sgt. Tony Ambrosio, Social Worker, Examiner.

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a research project being conducted by the Medical Department. They were further instructed that the results were to be used for later comparison with those of other soldiers taking the same test. Confidentiality was assured. A clinical psychology technician, using a standard set of rules extrapolated from Murray,[1] administered to each subject the Thematic Apperception Test. The only identifying material requested at that time was the subject's name and serial number. Afterwards, the social worker technician administered the check list and the information blank to the soldiers.

TABLE 1. *Description of 191 U. S. soldiers selected at random from the 35th Infantry Regiment*

Category	Percent	Category	Percent
Age		Religion	
18-21	60	Protestant	62
22-25	38	Catholic	35
26 and over	2	Other	3
Rank		Marital status	
Sergeant	8	Married	26
Corporal	15	Single	74
Private	77		
Race		A. F. Q. T.* score	
Caucasian	88	89 and under	37
Negro	9	90-109	48
Other	3	110-119	10
		120 and over	5
Education (years)		Combat time (months)	
0-6	7	1	1
7-9	27	2	11
10-12	52	3	8
13 and over	14	4	6
		0	74

*Armed Forces Qualification Test

The definitions for each variable were extracted from the extensive work of Murray. A short summary of each definition is offered in the following paragraphs. However, for the more extensive definitions used in this study, one should refer to *Explorations in Personality*.[2]

1. *Aggression.* To overcome opposition forcefully; to fight; to avenge an injury; to attack, injure, or kill another; to oppose forcefully or punish another.

2. *Abasement.* To submit passively to external forces; to accept injury, blame, criticism, punishment; to surrender; to become resigned to fate; to admit inferiority.

3. *Affiliation.* To draw near; enjoyably to co-operate or reciprocate with an allied object, an object that the subject likes or that resembles the subject; to please and win affection.

4. *Escape* (harm and blame avoidance). To avoid pain, physical injury, illness and death, humiliation, shame, ridicule; to avoid failure.

5. *Sexual misidentification.* To misidentify the sex of anyone pictured in the stories using McFarland's Criteria.[3]

6. *Constriction.* Measured by the total number of words used by the subject in telling the stories.

In variables 1 to 4, the direction and central object of the story was of little importance in determining the score. Aggression, however, was further broken down into medium of expression and directed object. Intensity was not scored in the analysis. A score of 1 was given to the subject if a particular variable was detected in a story. As 10 cards were used, the range of frequency scores thus varied from 0 to 10.

In order to ascertain the reliability of the examiner's judgment, an outside judge analyzed 37 records at random and applied the same set of rules. The percent of agreement between the two raters ranged from a low of 85.9 for verbal aggression directed toward the male to a high of 99.7 for sexual misidentification. These data amply indicate that the scoring rules for analysis are reliable as compared to acceptable standards in the research literature.

In order to check on all factors which might conceivably affect the tested relationships, personnel data characteristics were compared according to quartile distribution. With the exception of rank and combat time in the fourth quartile, there were no outstanding factors which affected the results in one direction or the other. In the former instance, one would expect a larger number of sergeants with more time in service. In the latter, the minimal period spent in a relatively quiet period of combat one year from the time of testing should have little influence on the experimental results.

Another pertinent factor that was taken into consideration was the time spent by the enlisted man in his individual unit. Thus, a soldier's emotional status may be conceivably affected by the duration of opportunity to identify with the group. However, though not statistically verified, the personnel policy in effect at the time of examination was one in which a man remained in his unit until rotated to the zone of the interior.

In reference to the statistical methodology involved, the total group was first broken down into four subgroups. The first quar-

tile represents the men who had completed up to 25 percent of their tours in Korea, whereas the fourth quartile represents the men who completed 75 percent or more of their present duty assignments. The average frequency score was then tabulated for each variable within the subgroup. This score was translated into a percent of the theoretical maximum score. Chi squares were applied to all measurements, with the exception of constriction and the psychosomatic check-list score. In these two latter cases, because total mean scores were utilized, a standard error measurement of the difference was applied as an index of statistical significance. The five percent level of significance was adopted in all cases.

RESULTS

Although the principal finding, as shown by table 2, is that there is no significant association between the variables tested and length of tour in Korea, some exceptions can be noted. In the case of the variable, aggression, the scores of the fourth quartile are significantly different from those of the second. On

TABLE 2. *Percent of frequency scores by quartile distributions for 191 U. S. soldiers**

Variables	Quartiles (percent of tour completed)			
	1	2	3	4
Thematic Apperception Test				
Physical aggression	19	18	21	22
Nondirected	4	5	5	4
Against male	8	9	10	11
Against female	5	4[1]	5	8
Against male and female	2	0	0	0
Verbal aggression	8	6[1]	8	10
Nondirected	2	11	2	1
Against male	2	2	2	3
Against female	1	1	1	3
Against male and female	2	1	1	3
Total aggression	27	24[1]	29	32
Affiliation	32	34[2]	29	27
Abasement	19	19	22	21
Escape	21	18	17	18
Sexual misidentification	8	5	7	7
Constriction**	444	479	515	473
Psychosomatic index**	8.4	7.9	8.3	7.7

*Percent of the theoretical obtainable maximum score.
**Values tabulated by means
1=2 quartile significantly less than 4 quartile.
2=2 quartile significantly greater than 4 quartile.

further inspection of this particular factor, the verbal medium of expression, in contrast to the physical variable, seems to maintain this relationship. However, the importance of physical aggression directed toward female objects as a significant component of this relationship is also evident. In contrast to the above, the variable of affiliation reaches its peak at the second quartile and is significantly different from its low at the fourth.

DISCUSSION

The major finding of this study is that there is no association between the majority of variables tested and length of tour in Korea. Thus, such factors as sexual identification, escape tendencies, abasement, and constriction, as well as psychosomatic complaints, remain relatively constant throughout the enlisted man's tour in Korea. Seemingly, therefore, one could infer a reasonable degree of resiliency in the U. S. soldier in warding off the emotional hazards of field duty in Korea.

Some exceptions to the above statement should be noted. The results seemed to indicate that as the soldier approaches the end of his tour in Korea, aggressive impulses become very much a part of his personality make-up when compared with his earlier adjustment. On the other hand, his need to draw close to a person, to feel companionship and friendship, becomes significantly lessened as the tour progresses. It may be hypothesized that the frustrations of overseas duties under field conditions that entail separation from home, family, and security have contributed to a reactive type of hostility and a withdrawal from social interaction. The importance of the female component as a focus for the hostility also seems to indicate an unconscious reaction to the prolonged separation from a normal heterosexual relationship.

Apparently these emotional forces are at their low ebb during the second quarter of the soldier's tour, when he is girding himself for the long pull ahead and requires an identification with his fellow workers. It must be noted, however, that though these presses and strains seem to exist, the data cannot ascertain the degree to which inner tension is converted into action. Finally, because there is no statistical difference between the third and second or fourth quartiles, though it is indeed an intermediate value between the two, these implications can be seen as being only suggestive.

SUMMARY

One hundred and ninety-one U. S. soldiers from the 35th Infantry Regiment were administered a psychosomatic check list and the Thematic Apperception Test. Over-all personality characteristics, as measured by the experimental tools, did not seem to be markedly affected by the 16-month tour of duty. The factors

of sexual identification, abasement, escape tendencies, and constriction remained relatively constant. There was some suggestion, however, both of a general increase in the soldier's aggressive impulses and of a decrease in affiliation needs as the tour progressed.

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PROBLEMS IN THE SERVICES

Men who are dependent and feel emotionally insecure are placed under severe conflictual stress if their wives verbalize dissatisfaction with Army life and suggest that their husbands terminate a career in which the men have made a large investment of time and have established good efficiency records.

Older men who are assigned markedly new duties for the first time in an overseas command and are required to adapt to an entirely new unit as individual replacements, while separated from their families, are likely to develop anxiety.

—FREDERICK A. ZEHRER, Lt. Col., MC, USA
in *Medical Bulletin of the U. S. Army, Europe,* p. 49, Feb. 1956

Late Complication of the Whipple Operation

Carcinoma of the Duct of Wirsung With Four-Year Survival

EDWARD J. FADELL, *Major, MC, USA*
MAURICE TULIN, *Major, MC, USA*

RADICAL pancreaticoduodenectomy for tumors in and about the head of the pancreas has fallen somewhat into disrepute in recent years. Of the problems arising from this procedure, two have received considerable attention: (1) the proper method for securing internal biliary tract drainage, and (2) whether the pancreatic stump should be closed or whether some form of pancreatico-enteric anastomosis should be attempted.[1]

We recently performed an autopsy on a previously reported case.[2] This report described an unusual tumor, a papillary adenocarcinoma of the duct of Wirsung which presented as a lemon-sized mass lying in the third portion of the duodenum and which was attached by a stalk to a point of origin in the terminal centimeter of the main pancreatic duct. No ampulla of Vater was present; the common bile duct and each pancreatic duct opened separately into the duodenum. The authors stated that they had been unable to find a report of a similar case in the literature. A pancreaticoduodenectomy was performed with direct biliary tract drainage by means of a choledochojejunostomy. The pancreatic stump was ligated and buried in the retroperitoneal tissues.

Because of the striking autopsy findings and also because of the rarity of the original tumor, we wish to report this follow-up.

CASE REPORT

History. This 58-year-old man was admitted to this hospital on 26 May 1954 with a diagnosis of carcinoma. Starting in 1945 he had recurrent episodes of jaundice, chills, and fever. An exploratory laparotomy was performed in 1948, and the gall bladder was removed. A common duct was found dilated at that time. No stones were present. The patient continued to have recurrent episodes of jaundice, chills, and fever, and re-exploration was done 1 December 1950. At that time a papillary adenocarcinoma of the distal 1 cm of the main pancreatic duct was demonstrated. It was connected to this primary site by a small pedicle, but the main portion of the tumor was a 5- by 6-cm mass in the

From Letterman Army Hospital, San Francisco, Calif.

duodenum. A modified Whipple operation was accomplished with removal of two thirds of the stomach, all of the duodenum, and the greater portion of the pancreas. The proximal end of the jejunum was closed and the common bile duct was anastomosed to it through a stab wound. A portion of the small intestine was anastomosed to the remaining portion of the stomach. The remaining portion of the pancreas was ligated and buried in the retroperitoneal tissue. The patient had a stormy postoperative course, during which time the pancreatic ferments drained through the anterior abdominal wall for a period of six months. Following discharge, the patient did fairly well until May 1954. At that time he began to have episodes of chills and fever. His appetite became very poor, and he was admitted to a local hospital, with transfer to this hospital on 26 May 1954.

Physical Examination. Physical examination on admission to this hospital revealed a chronically ill, cachectic white male, who appeared *in extremis*. Blood pressure was 70/40 mm Hg; pulse, 100 and regular. The chest was clear to auscultation and percussion. There was an indistinct, hard, diffuse, palpable mass in the upper part of the abdomen below the previous operative scars. The liver, palpable three to four fingersbreadth below the right costal margin, was moderately firm and tender. There was pigmentation of the skin; at the time of initial examination, it was not definite if this was jaundice.

Laboratory Data. Admission white blood cell count was 18,800 per μl with 95 per cent neutrophils, 30 of which were nonsegmented. The hemoglobin was 9.7 g/100 ml. Urinalyses were essentially negative. Serum bilirubin on admission was 2.1 mg (direct) and 3.6 mg (indirect) per 100 ml; four days later it was 2.4 (direct) and 4.6 (indirect). The serum bilirubin was not measured again but jaundice increased rapidly following these estimations. The serum amylase was 34 Somogyi units per 100 ml, and the serum lipase was 35 tributyrin units. The glucose, chlorides, sodium, potassium, and calcium levels were all within normal limits. The nonprotein nitrogen was 35 mg per 100 ml. The thymol turbidity was 19 units; thymol flocculation, 2 plus; zinc sulfate turbidity, 24; and cephalin-cholesterol flocculation, 3 plus in 24 hours and 3 plus in 48 hours. Two blood cultures were positive for *Escherichia coli*, which was sensitive to Aureomycin (brand of chlortetracycline hydrochloride). The clinical impression was metastatic carcinoma of the liver.

Clinical Course. On admission the patient was placed on a select diet, multiple small feedings between meals, 1 ml of vitamin B complex given intramuscularly daily, 360 mg (600,000 units) of penicillin twice a day, 1,000 ml of whole blood, 25 g of serum albumin, 50 mg of cortisone every 6 hours, and 25 mg of testosterone propionate intramuscularly twice weekly. Pancreatin after each meal was given. At first the patient improved slightly but spiked a temperature of about 102°F every second or third day. He then followed a gradual downhill course. Fever

was continuous and the jaundice increased greatly. Tube and intravenous feedings were resorted to without any improvement. The patient died on the morning of 10 June 1954.

Autopsy Findings

Gross examination. Examination of the body at autopsy showed a markedly jaundiced adult male who was well developed but poorly nourished. Upon opening the abdomen, an antecolic anastomosis was present between the proximal jejunum and the free border of the stomach. The pancreatic stump was buried in the retroperitoneal tissues. A choledochojejunostomy was present. Dissection of the anastomosis of the jejunum to the common bile duct revealed a large, mixed, pigmented stone measuring approximately 4 by 3 by 2 cm blocking the common duct. The liver weighed 2,000 grams. Sectioning throughout the liver revealed scattered nodularity due to abscesses. Green purulent fluid could be expressed. A pronounced nutmeg appearance was evident. The lungs showed numerous infarcts.

Microscopic examination. Microscopically the liver showed pronounced centrilobular necrosis. In many of the portal areas, abscesses (fig. 1) were present with a surrounding proliferation of granulation

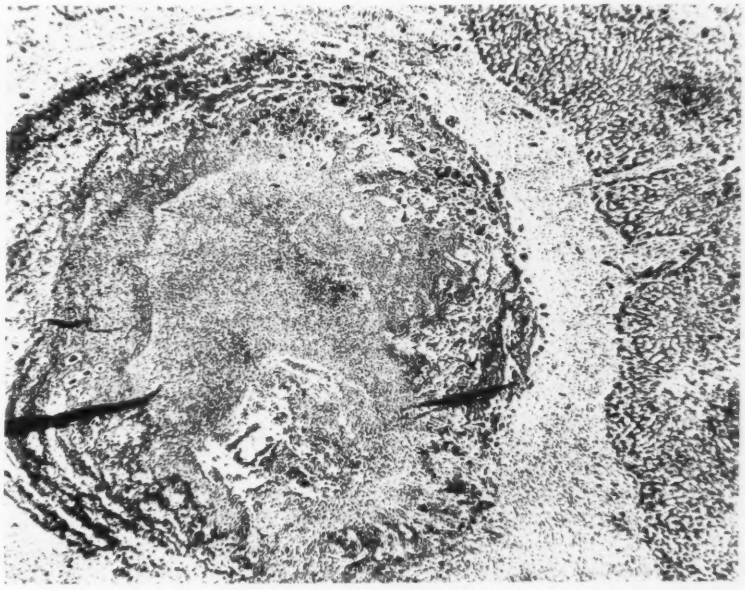

Figure 1. Portal abscess. (× 32)

tissue. In some portal areas a moderate proliferation of rather dense, mature, fibrous connective tissue was seen, together with a prolifer-

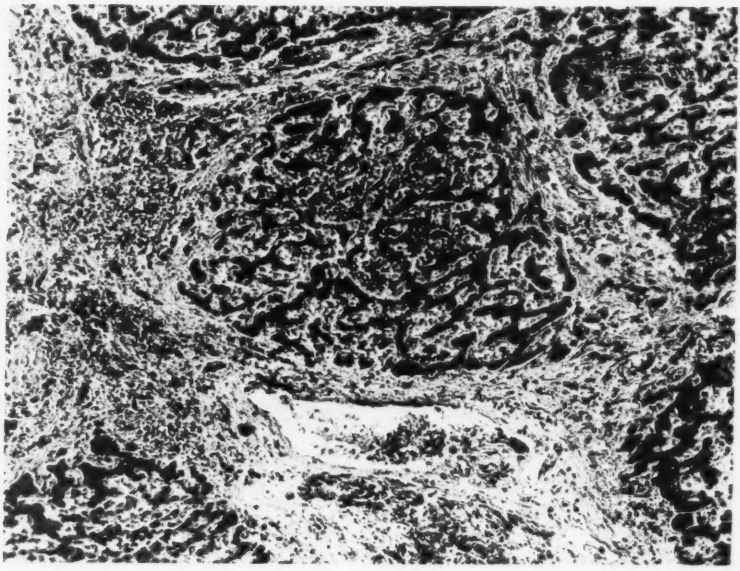

Figure 2. Portal fibrosis. (× 100)

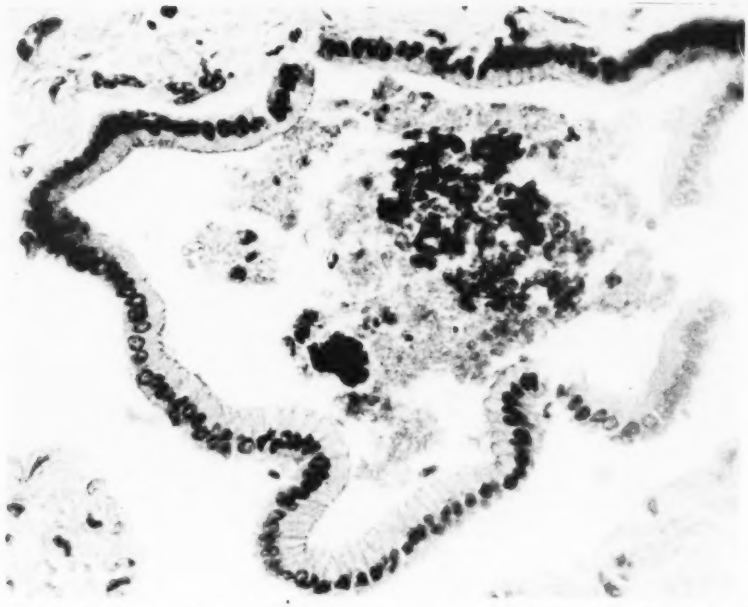

Figure 3. Liver. Dilated interlobular bile duct containing bile, debris, and leukocytes. (× 475)

ation of small bile ducts (fig. 2). Occasional bile ducts contained bile, debris, and leukocytes (fig. 3). Only occasional canaliculi contained bile thrombi. The pancreas revealed a pronounced loss of acinar tissue, with only islands remaining in the small portion of the pancreas left behind at the previous operation (fig. 4). There was no evidence of residual adenocarcinoma. The lungs showed areas of infarction, together with evident lobular bronchopneumonia.

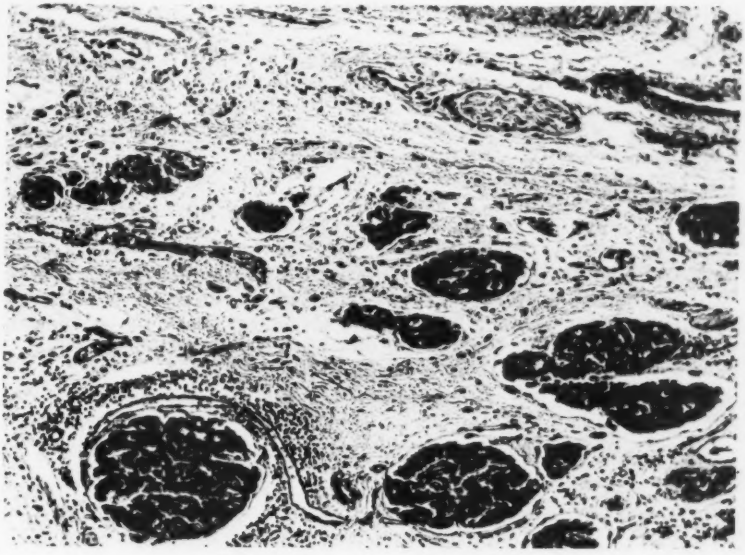

Figure 4. Pancreas. Residual island tissue and loss of acinar structures. (× 100)

Final anatomic diagnoses were:

1. Pancreatectomy, partial
2. Choledochojejunostomy
3. Choledocholithiasis with obstruction of the common bile duct
4. Abscesses, liver (secondary to ascending cholangitis)
5. Portal fibrosis (biliary cirrhosis, inactive), moderate, liver

DISCUSSION

Although the specimen resected at the operation contained tumor-infiltrated pancreas, duodenum, and lymph nodes, diligent search at autopsy revealed no tumor. A three and one-half year cure of cancer was, therefore, achieved.

A significant point of this case concerns a complication of the Whipple procedure; namely, calculus formation with cholangitis and consequent hepatic abscesses. Child and Ellis[1] described a similar complication in some of the few cases which were followed to autopsy after the Whipple procedure. Although the long-

followed cases are few, there is some degree of uniformity about the complications which suggests that the procedure, both as regards failure of eradication of the majority of cancers for which it is performed and the serious postoperative complications, may not be justified.

SUMMARY

The final autopsy report on a previously reported case of adenocarcinoma of the duct of Wirsung is submitted. Death occurred three and one-half years after removal of the tumor by a Whipple operation.

The terminal illness lasted approximately a month and consisted of anorexia; intermittent, remittent fever; increasing jaundice; evidence of moderate parenchymal hepatitis; and *Esch. coli* septicemia.

At autopsy, a large common duct stone and multiple hepatic abscesses were demonstrated. No evidence of residual tumor could be discovered. The report illustrates the probable cure of a pancreatic carcinoma and also the complications of the Whipple procedure.

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THE PROBLEMS OF A PHYSICIAN

"The young doctor may be disconcerted, on his entry into the professional world, to find malingerers among his patients. But he will find them. He will find hysterical patients, and gentle, amiable men and women who cultivate their neuroses as carefully as roses and cucumbers are cultivated for a horticultural show. And how is he to deal with them? The textbooks supply only part of the answer, and to complete it he must use moral courage: moral courage and perhaps ingenuity."

—ERIC LINKLATER
in *British Medical Journal*
p. 1519, Dec. 24, 1955

Causalgia in the Inguinogenital Area

EDWIN E. GOLDBERG, *Captain, USAF (MC)*
RICHARD D. AMELAR, *Captain, USAF (MC)*

CAUSALGIA is a term used to define a severe burning pain and hyperalgesia in the area of distribution of a particular nerve following partial injury to that nerve. The term was first defined by Mitchell, Morehouse, and Keen,[1] who in 1864 described its occurrence in soldiers following penetrating wounds. The exciting factor is usually direct trauma in the region of distribution of the nerve, but the condition may be precipitated by other irritants such as burns, adhesions, or operation scars; sometimes no causative factor can be determined. The sensory phenomena vary from an inconsequential mild burning sensation to an excruciating agony. The pain is constant, but is frequently exacerbated by movement of the affected part. Emotion may increase the severity of the pain. The pain is usually limited to, and most intense in, the sensory distribution of the involved nerve. The skin in the affected area may be normal, but frequently there are changes indicative of sympathetic nerve involvement, such as dryness, glossiness, scaliness, hyperhydrosis, coldness, redness, cyanosis, and other trophic disturbances.

Because causalgia is in some cases purely a manifestation of dysfunction of the sympathetic nerves, Wan,[2] in 1946, introduced the term "sympathetic causalgia." The actual mechanism of pain in causalgia has not been fully elucidated, although in obvious cases there are demonstrable pathologic changes in the nerves carrying the principal sympathetic supply to the involved area. At the level of injury these sympathetic fibers "make artificial synaptic contacts" with somatic afferent nerve fibers. In long-standing and severe cases, degenerative changes within the sympathetic ganglion cells have been noted. Sympathectomy often produces dramatic relief.

In April 1942 Magee[3] described an apparently previously unrecognized syndrome which he called genitofemoral causalgia and which was "characterized by pain and paresthesia in the distribution of the genitofemoral nerve." Magee described seven cases which he saw within a 12-month period. All of the patients operated on (six of the seven) were relieved either by

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freeing the nerve from adhesions or by completely resecting the nerve. In some cases there were evidently dense adhesions, in others only "filmy bands." In 1943 Magee[4] reviewed his cases of genitofemoral causalgia and analyzed six cases in which resection of the nerve and freeing of adhesions was the only form of therapy employed. The chief features presented by these patients were: (1) pain centered in the region of the internal abdominal ring; (2) tenderness on pressure over this area and the inguinal canal; (3) hyperesthesia over the distribution of the genitofemoral nerve; and (4) pain on hyperextension of the thigh.

Magee[4] stressed the fact that freeing the nerve from adhesions may give only temporary relief and that resection of the nerve is the only assurance of permanent cure. The condition is much more common in males, is usually right-sided, and frequently follows appendectomy, although the type of incision used in the appendectomy is apparently not related per se to the occurrence of this syndrome. In his report Magee[4] stressed the clinical manifestations of genitofemoral causalgia, but stated that "pathological study of the nerves removed shows normal nerve or slight interstitial fibrosis but no careful study of this aspect has been made." In 1945 Lyon[5] reported three patients with genitofemoral causalgia who were completely relieved by resection of the genitofemoral nerve. In one of these cases a microscopic examination of the involved nerve showed intraneural fibrosis and patchy demyelination. Lyon stated that in none of his cases were adhesions particularly dense but in all three cases that he described the nerves were "quite taut," and he goes on to state that he was unable to demonstrate this degree of tension in the nerves of normal individuals not complaining of this syndrome who were examined at laparotomy for other conditions.

The sensory nerves that supply the inguinogenital region are the anterior cutaneous branches of the iliohypogastric nerve, the ilio-inguinal nerve, and the external spermatic branch of the genitofemoral nerve. Since the latter two appear to have been involved in the case herein presented, a brief description of their anatomy is warranted.

The ilio-inguinal nerve arises from the first lumbar nerve, emerges from the upper part of the lateral border of the psoas major, and crosses obliquely in front of the quadratus lumborum and the iliacus. It perforates the transversus abdominis near the anterior part of the iliac crest, then pierces the obliquus internus abdominis, accompanies the spermatic cord through the external inguinal ring, and is distributed to the skin of the upper and medial part of the thigh and to the skin over either the root of the penis and upper part of the scrotum in the male or the mons pubis and labium majus in the female. The genitofemoral nerve arises

from the first and second lumbar nerves. After passing obliquely through the substance of the psoas major, it emerges from its medial border, descends on the surface of the psoas major under the peritoneum, and divides into the external spermatic and lumboinguinal nerves, although occasionally these two nerves emerge separately through the substance of the psoas. The external spermatic nerve passes outward on the psoas major, and pierces the transversalis fascia or passes through the internal inguinal ring; it then descends behind the spermatic cord to supply the cremaster and the skin of the scrotum.

CASE REPORT

A 33-year-old married pilot was transferred from an arctic air base because of persistent severe pain in the region of the right groin and the right testicle. The referring diagnosis was "probable calculus in the right ureter."

The history obtained from the patient is that the initial onset of his present illness occurred on 31 December 1953 when he was seized with right flank pain, radiating down to the right groin and right testicle. He received no relief from Novocain (brand of procaine hydrochloride) injections into the back muscles or from diathermy treatments, and an intravenous pyelogram performed on 2 January 1954 revealed no abnormalities of the upper urinary tracts. Following this, the patient stated, he was "packed in ice" for two days with some relief of his complaints and then put at rest in his quarters for a period of three weeks. Subsequently, he suffered from recurrent, severe, aching sensations in the region of the right testicle and occasional right-sided groin pains, more pronounced toward the end of the day; the scrotal pain was aggravated when the patient sat with his legs crossed. He went on 30 days' leave in November 1954, and on three separate occasions he noted severe right testicular pain which commenced during intercourse with his wife, so much so that he made no further attempts at coitus. A 200-mile bus ride on 18 December resulted in his having severe back pain that night. Prior to this time the patient had had no urinary symptoms. There was no urethral discharge, chills, fever, nausea, or vomiting. On questioning, he stated that straining at stool occasionally would cause right testicular pain. He continued to complain of pain in the region of the right testicle and groin and was referred to this hospital for further urologic evaluation and treatment. His past history was significant only in that he had an appendectomy for a ruptured appendix in 1936, at which time he was hospitalized for a period of 32 days.

Physical examination on admission to this hospital on 1 March 1955 revealed the patient to be lying flat in bed in moderate distress complaining of pain in the right testicle. He was alert and co-operative; his abdomen was flat and soft. The liver, spleen, and kidneys were not palpable. There was no costovertebral-angle tenderness. There was slight tenderness to pressure in the right inguinal region, but no hernia

or lymphadenopathy was detectable in the recumbent or upright positions. There were no skin changes in the area of the right groin. He had an irregular right rectus appendectomy scar which was well healed. Examination of the genitalia revealed a normal male complement with circumcised penis. The left testis, epididymis, and spermatic cord felt normal and were not sensitive to mild pressure. The right testis, epididymis, and cord felt normal, but there was tenderness to light pressure over the testis anteriorly and in the region of the epididymis. There was no induration or enlargement of the epididymis. On rectal examination, the prostate gland was felt to be enlarged 1 plus, symmetrical, somewhat boggy, and extremely tender to touch. Pressure on the prostate gland produced rather sharp pains deep in the perineum and in the suprapubic region. The seminal vesicles were not palpable or tender.

Prostatic massage, which was very painful to the patient, produced a thick, white mucoid discharge which on microscopic examination revealed only an occasional pus cell and a few gram-positive cocci. A postmassage, voided urine specimen was essentially normal with a scant amorphic sediment and an occasional white blood cell. The prostatic culture grew *Micrococcus pyogenes* var. *aureus* and *Streptococcus viridans,* sensitive to bacitracin and dihydrostreptomycin and faintly sensitive to Chloromycetin (brand of chloramphenicol), but resistant to Terramycin (brand of oxytetracycline), Aureomycin (brand of chlortetracycline hydrochloride), penicillin, and polymyxin. The voided urine culture grew the same organisms with the same sensitivities. Admission diagnosis was exacerbation of chronic nongonococcic prostatitis. Complete blood count was unremarkable and the sedimentation rate was 6 mm per hour. An intravenous pyelogram revealed normal upper urinary tract function and architecture bilaterally with no evidence of obstruction.

The patient was confined to bed and given Sitz baths and 100 mg of Furadantin (brand of nitrofurantoin) four times a day. He improved slightly on this regimen but after a week of hospitalization again complained of severe pain in the right groin. Examination revealed no objective signs of inflammation in the area. There was no fullness in the groin; the right scrotal contents appeared and felt normal, although they were tender to touch. These complaints continued and another prostatic massage was performed on 13 March without relief. On 17 March there was still no change in his complaints of persistent right groin pain. This pain apparently was precipitated or aggravated by standing, walking, or lifting, was of a burning nature, and seemed to originate in the groin and travel down into the scrotum. By this time he had had an adequate course of Furadantin (100 mg 4 times daily for 13 days) and streptomycin sulfate (1 gram daily for 10 days).

At this point it was felt that the patient's complaints, if due to a low-grade prostatitis, should have completely cleared up on this regi-

men. Since he had had almost no relief of his complaints, it was thought that his prostatitis was not producing his present symptoms. This raised the question of the possibility of the patient's condition being due either to a causalgia of the nerves supplying the right groin and scrotum or to a psychogenic disturbance. It was felt that causalgia involving the terminal branches of the ilio-inguinal and external spermatic portion of the genitofemoral nerves was possibly the cause of his symptoms.

On 18 March these nerves were blocked by infiltration with 1 per cent Novocain in a fan-shaped area extending from the right anterior superior iliac spine to an area 3 cm medial to this. This procedure gave the patient almost immediate relief of his complaints which lasted for approximately one and one-half hours. On 21 March, under intravenous pentothal anesthesia, the ilio-inguinal and genitofemoral nerves were severed at the region of the internal inguinal ring, and a two-inch segment of each nerve was removed. These specimens were individually marked and sent to the First Army Pathology Center for examination. The pathologist's* report indicated that "microscopic examination of these specimens show only sections of peripheral nerves with no diagnostic abnormality noted."

Following the operation the patient had no further testicular pain but complained of pain in the region of his inguinal incision for several days. However, he stated that this pain was different from the preceding pain that he had had, and it was believed to be due to the incision. Ten days postoperatively his only complaint was that of an occasional numb sensation in the area of the right testicle. He had no urinary complaints. A small area of anesthesia to touch was noted on the lateral aspect of the right scrotum and on the adjacent area of the inner aspect of the right thigh. The patient was anxious to resume his duties as a pilot, and he was returned to full duty.

He has been seen at two-month intervals since that time, and at the latest examination, in August 1955, he was feeling completely well, had no complaints, and was performing all of his duties. The area of anesthesia on the right thigh had disappeared, but he still had a small area of hypoalgesia on the lateral aspect of the right scrotum.

DISCUSSION

This patient suffered from causalgia of the right ilio-inguinal nerve and external spermatic branch of the genitofemoral nerve which was completely alleviated by section of these nerves high in the inguinal canal where they joined the spermatic cord. This is confirmed by his complete lack of symptoms since the operation was performed 12 months ago.

Chronic prostatitis may give rise to symptoms of pain referred to areas of the body supplied by spinal sensory nerve fibers

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arising from the tenth dorsal to the third sacral segments. The groin and genitalia are commonly involved by prostatic referred pain, and the physician is likely to indict the prostate as causing the pain, especially when the prostate gland is boggy or enlarged. In this case, the symptoms persisted in spite of adequate therapy for the prostatitis, and it was only then that our attention was drawn to the true diagnosis. The incidence of chronic prostatitis is high, especially in the military, and it is not unusual to have this disease concomitantly with other afflictions.

It is interesting to speculate on the importance of the past history of appendicitis as a causative factor in causalgia of the groin and genital region. On review of the literature we find that five out of seven of Magee's[3,4] cases and all three of Lyon's cases had previously had appendectomies. The relationship of this syndrome to previous appendectomy cannot be overlooked. It may be speculated that there is a perineural fibrosis involving the ilio-inguinal nerve in the region of the scar, but in our case we found no dense adhesions around the resected nerves, and the pathologist noted no anatomic changes. Possibly in some of the reported cases the appendix may have been removed because of a mistaken diagnosis when the causalgia was actually producing the pain.

It is hoped that, by reporting this case, attention can once again be drawn to this syndrome which, to the best of our knowledge, has been overlooked in the American literature.

SUMMARY

A review of the literature on causalgia of the ilio-inguinal and genitofemoral nerves is presented. An illustrative case report of an individual who was completely cured by section of these nerves high in the inguinal region is included. It is hoped that this report will stimulate consideration of this syndrome in the differential diagnosis of inguinal and genital pain.

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A MESSAGE FROM THE A. M. A.

Did you know the American Medical Association has a Bureau of Investigation? If so, are you familiar with its purpose and functions? To better acquaint military physicians with the activities of the A. M. A., the message this month is devoted to an answer of these questions.

The primary purpose of the A. M. A. Bureau of Investigation is the collection and dissemination of information on "patent medicines," quacks, medical fads and various other phases of pseudo-medicine. Since 1906 the Bureau, one of the educational activities of the Association, has served as a clearing house for information on the subjects with which it deals.

In the earliest days, the Bureau of Investigation was known as the Propaganda Department. Expository articles published in the A. M. A. Journal were compiled for the first time in 1912 and became the first volume of *Nostrums and Quackery*. A companion publication, devoted also to drugs promoted to the medical profession, called, "Propaganda for Reform," was published in 1917 by the Council on Pharmacy and Chemistry. It utilized many Bureau reports. The second volume of *Nostrums and Quackery* was published in 1921, and Volume Three appeared in 1936.

As one can well imagine, the reports of the Bureau were not popular with those immediately affected thereby. Naturally, a good many uncomplimentary things were said. As a result, no less than 42 law suits were filed against the Association for libel. The sums claimed as damages totaled over $24,000,000. A good many of these cases were perhaps filed only for publicity purposes, and were never tried. A few, however, were fought to the conclusion, and the Association was successful in every instance except one. A Bureau report had mentioned the fact that the promoter of an alcoholic "tonic" on weekdays was a pillar of the church, preaching temperance on Sundays, and the jury felt that this was perhaps a little too harsh on the man and awarded him the sum of one cent. In other notable cases the Bureau accused widely known figures of being quacks and then proceeded to prove the characterization.

Information on over-the-counter medicines and such related subjects as devices, food faddists, cultists, and quacks is available to physicians, their patients, students and educators, government agencies, and civic groups. Federal, state, and munic-

From the Council on National Defense of the American Medical Association. The views and opinions expressed are not necessarily those of the Department of Defense. —*Editor.*

ipal health officials, physicians, and others interested in public health often refer inquiries to the Bureau. In preparing news and feature stories, newspapers and magazines frequently rely upon information in Bureau files.

Information is collected by the Bureau from several sources. Original investigations, often supplemented by analytical work done in the A. M A. Chemical Laboratory, are carried on. Data are received from federal sources such as the Food and Drug Administration, National Institutes of Health, Post Office Department, and Federal Trade Commission, as well as from state licensing boards and state and municipal boards of health. Information also is gleaned from reports in domestic and foreign technical journals and from reports of special committees. Doctors of medicine and local and state medical societies are valuable sources of information.

Thousands of inquiries are received by the Bureau each year. The Bureau transmits its information mainly through replies to these letters. Articles are prepared by the Bureau for the *Journal of the American Medical Association* and for *Today's Health,* which may later be reprinted with permission in pamphlets and books. A physician in need of material for lectures on quacks and nostrums may wish to contact the Bureau.

Although in past years the Bureau published a number of books and pamphlets on nostrums and quackery, the passage of the 1938 Federal Food, Drug, and Cosmetic Act requiring the accurate labeling of drugs has reduced the need for these publications. Reprints on many of these subjects still are available.

In addition to its principal function of furnishing what information it has in its files on specific request, the personnel of the Bureau spends a good deal of time and effort in the exposure of medical impostors. There have been times when rather clever individuals have posed as doctors under circumstances which provided a real challenge, born either of their ingenuity or their influence in certain circles. In one case an extraordinary amount of effort over a period of about four years resulted in the exposure and "unfrocking" of a man who had posed as a Doctor of Medicine in a southwestern state for 20 years.

The Bureau has available to medical groups and educators lantern slides and a film strip on nostrums and quackery. Many newspapers and magazines call on the Bureau for information regarding advertising. The National Better Business Bureau and affiliated local Better Business Bureaus are in close co-operation with the A. M. A. Bureau. Large business organizations sometimes call on the Bureau for information on medical devices and products which may be offered to their employees.

FAMOUS SPECIALIST AND SURGEON GENERAL OF NAVY VISIT WEST PACIFIC

Rear Admiral Bartholomew W. Hogan, MC, USN, Surgeon General of the Navy, and Dr. Paul Dudley White, consulting physician to President Eisenhower, made a tour of naval medical facilities in the Pacific, returning to the United States late in April. Other members of the party included Captain Walter Welham, MC, USN, Staff Medical Officer, Commander Submarine Forces Pacific, and Captain Leona Jackson, NC, USN, Director of the Navy Nurse Corps. Captain Charles W. Stelle, MC, USN, Staff Medical Officer, Commander Naval Forces Far East, accompanied the group during visits to activities in that command.

Dr. Paul Dudley White and Rear Admiral Bartholomew W. Hogan congratulate four Japanese doctors on having successfully completed a one-year internship at the U. S. Naval Hospital, Yokosuka, Japan. The interns are, from left to right, Dr. Yutaka Katoh, Dr. Noboru Tokumaru, Dr. Yooichiro Yanagita, and Dr. Kooichi Fujii. Drs. Katoh, Yanagita, and Fujii will continue their training in Maryland, Johns Hopkins, and Washington University hospitals respectively, beginning in June or July.

The trip covered installations in Japan, Okinawa, Formosa, the Philippines, the Marianas, Kwajalein, and the Hawaiian Islands. Dr. White spent several days at the Cardiology Research Unit in Fukuoka, Japan, which he is directing with Dr. Ancil Keyes, and spoke on cardiac diseases at a number of symposia and meetings for medical officers of our Armed Forces. He also consulted on many patients in military hospitals and dispensaries. In Manila, Admiral Hogan and Doctor White attended sessions concerned with the establishment of an Asian-Pacific Congress of Cardiology, in which Dr. White was greatly interested.

A friendly interlude during the survey of medical conditions and requirements on Saipan, Mariana Islands.

Admiral Hogan conferred with senior naval commanders and interviewed many busy medical officers at the activities that he visited, placing special emphasis on making the best possible use of the time of valuable doctors, who are in such short supply. Medical, dental, and nursing activities were observed throughout the trip and conferences were held on problems related to patient care and medical logistics. Particular attention was given to the feasibility and reasonableness of making consolidations without causing reduction of the highest standard of patient care.

REGULAR MEDICAL CORPS OFFICERS CERTIFIED BY SPECIALTY BOARDS

Supplementary Listing

According to current information from the Offices of the Surgeons General of the three military medical services, the following 329 officers have been certified by the boards indicated, since the listings published in successive issues of this *Journal* from October 1953 to April 1955.

The American Board of Otolaryngology

James P. Albrite, Maj., USA
Stanley H. Bear, Maj., USAF
Aubrey K. Brown, Lt. Col., USA
John J. Brown, Capt., USN
Lawson G. Cox, Maj., USA
Joseph B. Dominey, Jr., Comdr., USN
Byron T. Eberly, Lt. Comdr., USN
William C. Livingood, Capt., USN
Gordon Marquis, Maj., USAF
Bernard J. Moore, Lt. Col., USA
Francis J. Peisel, Maj., USA
Charles H. Ransom, Lt. Col., USA
Cecil D. Riggs, Capt., USN
Maurice Schiff, Comdr., USN
Herbert P. Sube, Lt. Col., USA
George W. Taylor, Jr., Comdr., USN
Gilbert A. Twitchell, Lt. Col., USAF

The American Board of Ophthalmology

William R. Armstrong, Lt. Col., USAF
Virgil A. Beuerman, Capt., USN
Charles O. Bruce, Jr., Maj., USA
Warren T. Culver, Maj., USAF
James L. Curtis, Maj., USAF
James L. Fuelling, Capt., USN
Robert Kelley, Col., USA
Theodore N. Kirkland, Jr., Lt. Comdr., USN
George A. Levi, Maj., USA
John W. Linfesty, Col., USAF
Harold J. Louis, Maj., USAF
Lemuel T. Moorman, Lt., USN
Jack W. Passmore, Lt. Col., USA
Charles O. Rixey, Lt. Col., USA
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FLAGG, Roger H., Lt., USN
FLYNN, Ralph F., Jr., Lt., USN
FOSTER, Richard D., Lt., USN
FOULKE, Charles R., Lt., USN
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HAGAN, Edmund H., Lt., USN
HALL, Samuel D., Jr., Lt., USN
HAMBLIN, John A., Lt., USN
HANSEN, Van Ness, Jr., Capt., USAF
HANGER, Herbert L., Lt., USN
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HENDRICKSON, Richard D., Lt., USN
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JOHNSON, Verland E., Capt., USAF
JUDD, Thomas W., Capt., USAF
KANE, Edwin W., Lt., USN
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KARLIN, Harold R., Lt., USN
KARMEL, Edwin H., Lt., USN
KELLER, Marion H., Jr., Lt., USN
KELLY, Robert B., Lt., USN
KESTY, Thomas J., Lt., USN
KIDLE, Elroy D., Lt., USN
KING, William E., Lt., USN
KIRBY, Robert G., Lt., USN
KLEIN, Sanford E., Lt., USN
KOHNKE, Robert J., Lt., USN
KOPPERUD, William H., Lt., USN
KRAUSE, James E., Lt., USN
KRAUSE, James H., Lt., USN
KUHN, Marvin M., Lt. Col., USA
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LIGGETT, James M., Lt., USN
LINDBLOM, Penner P., Lt., USN
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LUDWICK, Thomas E., Lt., USN
LUZZI, James M., Lt., USN
LYONS, David F., Lt., USN
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MATTHIAS, William T., Lt., USN
McCALL, Howard W., Lt. Col., USA
McCHESNEY, Warren J., Lt., USN
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MUIR, Richard J., Lt., USN
MURPHY, James H., Lt., USN
NORWOOD, William K., Jr., Lt., USN
O'BRIEN, Argyle C., Lt., USN
O'CONNER, John E., Lt., USN
O'FLAKAGAN, Thomas M., Lt., USN
OLSON, Arthur E., Capt., USAF
OSTERGAARD, Carl M., Lt., USN
OTT, William Y., Capt., USAF
OUTTEN, Joseph F., Capt., USAF
OWEN, Hulett B., Capt., USAF
PERCIFUL, Jack R., Lt., USN
PHILIPS, Constantine F., Lt., USN
PHILLIPS, Clark B., Lt., USN
PHILLIPS, Van A., Capt., USAF
PISTOCCO, Louis R., Lt., USN
PRANGE, William H., Lt., USN
PRUITT, Clarence C., Capt., USAF
QUIRK, George P., Lt., USN
REID, Albert F., Lt., USN
RICHARDSON, Benny C., Lt., USN
RICHTER, Marvin A., Lt., USN
ROBBINS, Austin, Lt., USN
ROBINSON, Charles R., Lt., USN
ROPER, Jack, Lt. Col., USA
ROSENBERG, Irving, Lt., USN
RUEDEMANN, Paul P., Lt., USN
SALINGER, Werner, Lt., USN
SALVI, Henry V., Capt., USAF
SAMKOFF, Max D., Lt., USN
SCANGARELLA, Joseph J., Capt., USAF
SCHABOWSKI, Anton T., Lt., USN
SCHMITT, Oscar A., Lt., USN
SCHNELL, Robert A., Lt., USN
SCHOLZ, Kenneth C., Capt., USAF
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SCHROETER, Paul W., Lt., USN
SCHWARZ, Eugene R., Lt., USN
SCIRCLE, Robert B., Lt., USN
SCOLNICK, Arthur M., Lt., USN
SCOTT, Edward A., Capt., USAF
SCULLY, John J., Jr., Lt., USN
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THOMPSON, James T., Lt. Col., USA
TOYE, James C., Lt., USN
TRAFELI, Mario M., Jr., Lt., USN
TRIBE, Dale L., Lt., USN
UDALL, Richard P., Lt., USN
VANEENENAAM, Robert D., Lt., USN
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VICKERS, Jack R., Lt., USN
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WHITE, Edward M., Lt., USN
WIAN, George I., Jr., Lt. Col., USA
WILLIAMS, James S., Lt., USN
WOODEN, Robert A., Lt., USN
WOOL, Arthur L., Lt., USN
YEAGER, Arthur L., Lt., USN
ZABKA, Clifford, Lt., USN
ZAZZARO, John J., Jr., Lt., USN
ZACK, Justin S., Lt. Col., USA
ZUCKERMAN, Julius, Lt., USN

Reviews of Recent Books

GLAUCOMA, A Symposium organized by The Council for International Organizations of Medical Sciences, established under the joint auspices of UNESCO and WHO. Editor, Sir *Stewart Duke-Elder,* England. 350 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $7.50.

This symposium, composed of 20 of the best-known international authorities on this subject, was extremely timely and necessary due to the recent advances in knowledge on glaucoma. This excellent book not only brings the subject of primary glaucoma up to date, but also attempts to standardize nomenclature. The purpose of the symposium is stated beautifully in a thought-provoking introduction by Sir Stewart Duke-Elder on the "Fundamental Problem of Glaucoma"—the etiology and fundamental nature of primary glaucoma.

Barkan, in 1938, introduced an anatomic method of nomenclature, and used the terms "narrow-angle" and "wide-angle" glaucōma. This was a step forward but still not ideal. Until more satisfactory terminology is proposed and accepted, the symposium has advocated the terms "closed-angle" and "simple" glaucoma.

Approximately half of this printed symposium is devoted to the anatomic, physiologic, and related aspects of primary glaucoma, the rest emphasizing gonioscopy and provocative tests, with least emphasis on the clinical aspects of therapy and treatment.

This is a most excellent and timely book on the fundamentals of glaucoma, made interesting by the comments of the various panel members, and showing by their occasional disagreement that the subject is still in the process of being studied, investigated, and becoming understood.

Due to the problems posed, the many unanswered questions, and the necessity to search out the causes of glaucoma, this symposium necessarily emphasizes the theoretic rather than the clinical aspects.

Considerable scientific and experimental physiologic data have been included and used as a basis for many of the theories promulgated by the symposium members. The scientific part is of great value for researchers and thorough students of the subject, yet the conclusions drawn from the research are presented in such a readable manner, both as scientific papers and as discussions by the clinical members of the panel, that not only the man in research and the advanced student, but even a busy practitioner with limited time to read, can profit by reading this symposium.—*KARL D. MacMILLAN, Col., MC, USA*

SUBACUTE BACTERIAL ENDOCARDITIS, by *Andrew Kerr, Jr.*, M. D. A Monograph in the Bannerstone Division of American Lectures in Internal Medicine, edited by *Roscoe L. Pullen*, M. D., F. A. C. P. 343 pages. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $6.50.

The author has condensed a wealth of material into a monograph of some 266 pages covering a subject of interest to everyone in the field of medicine and one which we agree was in need of documentation. It is just as true today as in 1885 when Sir William Asher said of ulcerative endocarditis, "It is of use from time to time to take stock, so to speak, of our knowledge of a particular disease, to see exactly where we stand in regard to it."

The chapters covering antibiotic therapy are outstanding, as is the bibliography. The chapter on the history of subacute bacterial endocarditis is particularly well done and makes interesting reading. In some places, however, the author has incorporated too much statistical data which makes the reading a bit difficult.

The monograph is excellent and can be recommended as good reading to those interested in this disease. It is recommended to the busy general practitioner as a splendid reference.

—*MELVILLE M. DRISKELL, Capt., MC, USN*

A COURSE IN PRACTICAL THERAPEUTICS, by *Martin Emil Rehfuss*, M. D., F. A. C. P., LL. D. (Hon.), *Alison Howe Price*, M. D., and 24 contributors. 3d edition. 972 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., 1956. Price $15.

This book presents a comprehensive course in clinical therapeutics by 24 contributors, all but two of whom are from the staff of Jefferson Medical College or Hospital. This adds greatly to the continuity of the text. Of necessity the text is voluminous and is not to be read from cover to cover but should be on the desk of every practicing physician for ready reference. It is ideal for interns and residents.

This edition has retained the same format of the previous two. It is written in outline form with a double column to each page for easy readability. It has been revised, and recent advances in medicine and technics have been included. The illustrations are of excellent quality and consist of schematic drawings and colored plates that are used mainly as diagnostic aids.

The first section on general therapeutic principles is short but covers such important subjects as diagnostic survey, formation of a definite plan of treatment, and advice as to contents of the physician's bag. In the next secion on symptomatic therapy, page references to one of the other two sections, the treatment of specific disorders, and special treatment, are given whenever indicated.

This book fulfills its intended purpose. It is well worth its cost and is recommended for the daily use of the practicing physician.

—*PATRICK I. McSHANE, Col., MC, USA*

ATOMIC ENERGY RESEARCH AT HARWELL, by *K. E. B. Jay.* 144 pages; illustrated. Philosophical Library, Inc., New York, N. Y., 1955. Price $4.75.

This book is a generalized summary of the functions of the Atomic Energy Research Establishment at Harwell in England. It is divided into two parts, the first of which is concerned with the Harwell establishment participation in various fields of research such as reactors, isotopes, instrument development, et cetera, and how these efforts are co-ordinated with and support the production and weapons development phases. The second part gives a detailed description from a fundamental research standpoint, of some important projects under study, which are carried on in the physics, chemistry and chemical engineering, and metallurgy divisions.

The work is readable and in pleasing style, but understanding it requires some knowledge of the general subjects covered and of atomic energy programs. The part on fundamental research presupposes a fairly extensive background in science as stated in the author's foreword to that section.

The entire text well outlines the scope of work being done at Harwell, with enough references to other agencies within the United Kingdom's atomic energy program, without getting into classified information, so that an over-all inference may be drawn as to their extensive coverage in the field. —*CLINTON S. MAUPIN, Col., MC, USA*

A. M. A. SCIENTIFIC EXHIBITS 1955. Sponsored by Council on Scientific Assembly, American Medical Association. 784 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., 1955. Price $20.

For the first time, physicians unable to attend the American Medical Association convention held in Chicago, 1955, will be able to "see" the exhibits presented and study them at their leisure.

The exhibits depicted in this volume approximate one third of the total shown at the Annual Convention. Many of the exhibits depicted in this volume have been singled out by the A. M. A. Committee of Awards for Certificates, Awards, and Honorable Mention. Included also are exhibits that have a wide appeal for practicing physicians. While the photographs depicting these exhibits mirror some of the shortcomings of the exhibits themselves, it must be remembered that they are not primarily constructed for the purpose of photographic reproduction. Further, it would not be practicable, even if possible, to construct exhibits to serve the dual purpose of "live" as well as photographic presentation. In addition, some of the photographs submitted by the exhibitors were nonprofessional in quality. To attempt to do such work at a professional level and in color would have been financially prohibitive.

For these reasons, the reader will discover, from time to time, a portion of a page on which graphs or copy appear faded or illegible.

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Some illustrations also suffer from the inferior quality of original photography submitted for reproduction. Despite these obvious shortcomings, common to any pioneering effort, this book should have a wide appeal to the many physicians who could not be present at the convention. There is no other book to which they can turn for this information, and the information presented is, in effect, a postgraduate course par excellence available to them nowhere else.

The planning and production of this volume of some 800 pages was a prodigious undertaking requiring rare courage from inception to delivery. The A. M. A. has a "lion by the tail," and this reviewer hopes that it won't let go.—*ROBERT V. SCHULTZ, Capt., MC, USN*

VIRUS AND RICKETTSIAL DISEASES, by *S. P. Bedson,* M. D., D. Sc., F. R. C. P., F. R. S.; *A. W. Downie,* D. Sc., M. D.; *F. O. MacCallum,* M. D.; and *C. H. Stuart-Harris,* M. D., F. R. C. P. 2d edition. 406 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., 1955. Price $6.75.

In this small book, an attempt is made to summarize for the "practicing physician" current knowledge of the virus and rickettsial infections of man. As in the first edition the text is written by four authors and is subdivided into chapters concerned with specific diseases. Each of the authors endeavors to present a coherent, readable account rather than an exhaustive treatise on the various diseases, and the space devoted to the various chapters is determined by extent of knowledge rather than the medical importance of the disease. Infections are described primarily from the causative point of view, and clinical descriptions are limited to bare outlines. Descriptions of procedure for laboratory diagnosis, while sketchy at times, are included wherever possible. Epidemiology and disease control are stressed where increased knowledge of the infectious agent and of its natural history has indicated measures of practical value.

In its broad outline, this second edition remains essentially unchanged. Most important revisions have been made of the chapters on poliomyelitis and the chemotherapy of rickettsial infections. A description of Murray Valley encephalitis has been added to the chapter on arthropod-borne virus encephalitis, and the Coxsackie viruses have been allocated a chapter of their own. These additions and revisions were prompted by recent advances in knowledge of these specific infections. The book retains its introductory chapters on the general characteristics, immunology, and natural history of viruses, but drops the older section on bacteriophage. Illustrations are well done and appropriate to the text.

This volume will serve as an excellent introductory text in medical virology for students or as a handy office reference for the practicing clinician. For the clinician, an outline of methods for laboratory diagnosis of virus and rickettsial disease is presented as an appendix. This outline is comprehensive yet concise and indicates the nature

of specimens required for laboratory diagnosis. This book, however, with its abridged text and shortened bibliography, is not sufficiently comprehensive to satisfy the scientifically curious physician or to be of much value to the student of infectious disease.

—*EDWARD L. BUESCHER, Maj., MC, USA*

HUMAN PHYSIOLOGY, by *F. R. Winton,* M. D., D. Sc., and *L. E. Bayliss,* Ph. D. 4th edition. 616 pages; 236 illustrations. Little, Brown & Co., Boston, Mass., 1955. Price $8.

This is the fourth edition of a now familiar small textbook on human physiology. A number of well-known English professors, currently active in research and teaching, have collaborated in a direct approach to the essential fundamentals. In order to keep the book within a readable size for the student, many of the nonessentials have been intentionally omitted. Nevertheless, the details, as presented, are sufficient to encourage "the intellectual attitude of an alert explorer."

This edition is divided into 17 sections, including several entirely new chapters. Those on respiration, body fluids, and transmission at nerve endings are set forth in a particularly clear and practical manner. Perhaps it is a little early for incorporation into a standard text, but I was surprised to note that there is little reference to recent advances in our understanding of physiologic principles through biophysical technics, including the use of isotopes.

The volume is written in the simple, direct fashion that we have come to admire in English textbooks. In addition to its use as a student's text, I would like to recommend it as a general reference for those interested in the biologic sciences who feel they require a review of the principles of human physiology.

—*JAMES B. HARTGERING, Lt. Col., MC, USA*

ATLAS OF GENERAL SURGERY, by *Joseph R. Wilder,* M. D. 222 pages; 101 plates. The C. V. Mosby Co., St. Louis, Mo., 1955. Price $13.50.

This book is an illustrated atlas of general surgery. The author, by the use of selected line drawings, describes the surgical technic in a large number of standard procedures most frequently performed by the general surgeon. The author has included only those procedures and technics that are generally accepted and has eliminated those which are controversial.

Preceding the description of the various steps in each operation is a brief discussion of the indications for the procedure, together with a list of special considerations, which mention the complications and dangers in the performance of the operation.

This book will be especially valuable as a reference book for the medical student, intern, resident, and general surgeon.

—*HAROLD G. YOUNG, Capt., MC, USN*

PEPTIC ULCER, Diagnosis and Treatment, by *Clifford J. Barborka,* M. D., M. S., D. Sc., F. A. C. P., and *E. Clinton Texter, Jr.,* M. D. 290 pages; 33 illustrations. Little, Brown & Co., Boston, Mass., 1955. Price $7.

This small handbook crams a tremendous amount of valuable information on peptic ulcer into its pages. The authors, who are recognized authorities on gastro-intestinal diseases, touch the high points of the ulcer problem lightly, but show great skill in sifting out what is of essential merit from the vast amount of medical literature that has accumulated on this disease. The work is essentially a compact, handy reference book for the general practitioner and medical student. It is not sufficiently comprehensive to serve as a source book for the gastroenterologist, research worker, or abdominal surgeon. It is written in an engaging and easily readable style. The latest developments on the etiology and pathogenesis of peptic ulcer and the current opinions of well-recognized gastroenterologists on the medical and surgical treatment of the disease are presented in a concise, authoritative manner. Much of the content is so clearly and simply presented that it will be intelligible to the educated lay ulcer patient, and will help him understand and manage his disease more intelligently.

The book is composed of 14 short chapters, each of which is followed by a fairly extensive bibliography. The chapters on symptomatology, diagnosis, medical treatment, dietary management, complications, and the gastric ulcer-cancer problem are especially well written. The chapters on anatomy and physiology, etiology, and pathogenesis are hampered by the necessity of condensing a large amount of material into a few pages. The authors' approach to the gastric-ulcer problem is rational and impartial. The importance of the doctor-patient relationship in the management of peptic ulcer is given proper emphasis, but the very important role of emotional factors in the etiology and management of ulcer is given too little consideration. The discussion of the anticholinergic drugs and their use in treating acute and chronic ulcer disease is well presented.

In summary, this book should prove to be a handy reference book for the busy general practitioner and medical student. Portions of it should be suitable for the ulcer patient who is interested in learning more about his disease.—*EMMETT L. KEHOE, Col., MC, USA*

A SURVEY OF CLINICAL PRACTICE IN PSYCHOLOGY, edited by *Eli A. Rubinstein,* Ph. D., and *Maurice Lorr,* Ph. D. 363 pages. International Universities Press, Inc., New York, N. Y., 1954. Price $6.

The versatility of situations in which clinical psychologists make useful contributions to the understanding of the problems of individual human beings is shown by 27 chapters, each devoted to a different area or setting. The impossibility of the individual psychologist's being conversant with the entire range of practice is patent. For instance, despite clinical experience with public school, Navy, county, medical school, and Veterans Administration facilities, the present

reviewer could claim personal acquaintanceship with only six of the 27 areas discussed. This book, therefore, can supply orientation and specific information for practicing psychologists who ought to be informed about clinical settings other than their own.

Illustrated by the various chapters are the tasks, methods of approach, tools used, and research challenges to be found in clinics for aged persons, alcoholics, students, employees; in clinics attached to municipal and juvenile courts, training schools for delinquents, and prisons; in clinics for speech problems, hearing problems, reading disability, and physical rehabilitation; and in medical school centers, psychological service centers, and private practice. There are also excellent descriptions of the relatively large scale (as compared with a university clinic, for example) programs for the utilization of the abilities of psychologists in the Veterans Administration, the Army, the Navy, the Air Force, and the Public Health Service. Even the psychologists actively working in these programs will be able to learn more about the place of their own professional responsibilities in the whole picture.

This is an exceptionally well-planned volume for coverage and for the competence of the individual contributors. It will be of great use to clinical psychologists on a professional level, but will also be used in the training of psychologists—not to tell them about how to test or to interview, but to tell them some very practical things about how it is to be a clinical psychologist earning a living and trying to make the world a little better and happier for those served by psychology.

—WALTER L. WILKINS, Comdr., MSC, USNR

TEXTBOOK OF SURGERY, edited by *H. F. Moseley,* M. A., D. M., M. Ch. (Oxon), F. A. C. S., F. R. C. S. (Eng.), F. R. C. S. (C). With Foreword by *G. Gavin Miller,* M. D., C. M., M. Sc., F. R. C. S. (C), F. A. C. S. 2d edition. 1,136 pages; with 571 illustrations and 79 color plates. The C. V. Mosby Co., St. Louis, Mo., 1955. Price $16.50.

The foreword states that the objective of this text is to supply the medical student with the surgical information which he needs in order to graduate. To comply with that objective is a task which leads to differences of opinion relative to the merit of this or that subject. What appears appropriate to one contributor may raise a storm of controversy from others.

The orderly arrangement of the contents makes the volume easy to use as a ready reference. Many of the chapters devote much space to surgical technic that is not within the province of the medical student but which may, however, be of value to residents and practitioners. In this respect, it may be construed that the text oversteps its stated objective and some of the contributions lead into highly controversial fields which can be truly evaluated only by experience.

The illustrations and color plates are interesting and instructive. Much credit should be given to those responsible for the illustrations.

The bibliographies and film references are very useful and the references to the teaching film libraries are particularly welcomed. The new chapters on radioactive isotopes and on the acute abdominal conditions have clearly brought out many timely advances.

The plan to place in one volume all that the medical student should know relating to general surgery and all the surgical specialties has resulted in the production of this first Canadian Textbook of Surgery. It is now placed alongside several other textbooks of similar nature, providing the student another choice for his personal library.

—*PHILIP J. McNAMARA, Capt., MC, USN*

INLAYS AND ABUTMENTS, Their Preparation and Construction for Dental Restorations, by *Jacob R. Schwartz*, D. D. S. 478 pages; 512 illustrations. Dental Items of Interest Publishing Co., Inc., Brooklyn, N. Y., 1953. Price $8.

The text is directed primarily to the student, although the excellent illustrations of the required steps for each cavity preparation should provide a valuable reference for the practitioner. The meticulous detail devoted to armamentarium, classification of cavities, and the operator's position at the chair will be appreciated by the student, but is likely to be tedious to the practitioner who is not interested in a review of these subjects.

The chapters follow in logical sequence with the exception of chapters 5 and 6. Chapter 6 on "Operative Positions at the Chair" would be better situated at the beginning of the text, and chapter 5, "A Mathematical Computation of the Permissible Number of Practical Preparations," could be omitted without altering the scientific value of the publication. Indications and contraindications for the various restorations described are insufficiently emphasized, and the very important phase of treatment plan with diagnostic aids has been completely neglected.

The chapters "Highlights in Dental Casting," "Casting Investment Compound," and "Controlled Casting" are worthy inclusions and could be advantageously perused by anyone engaged in the casting of gold.

The text is well written and contains many historical highlights of dentistry, as well as a thorough historical background for each restoration. The abundant illustrations are exceptionally well done and are judiciously dispersed to simplify the step-by-step progress of each cavity preparation.—*LESLIE R. ALLEN, Lt. Col., USAF (DC)*

AMERICAN PHARMACY, Textbook of Pharmaceutical Principles, Processes and Preparations, edited by *Rufus A. Lyman*, M. D., and *Joseph B. Sprowls*, Ph. D. Consulting Editor, *George Urdang*, Ph. G., D. Sc. Nat., Sc. D. (h. c.). 4th edition. 478 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., 1955. Price $9.75.

This well-known textbook is in its fourth edition in the past 10 years. The new edition brings it abreast of the latest revisions of the *U. S. Pharmacopeia* and the *National Formulary*. It is divided into two

parts, the first concerned with general principles and processes and the second with specific pharmaceutical preparations. In the first part, as is usual, a great deal of space is devoted to weights and measures and the elementary chemical and physical procedures used by the manufacturing pharmacist. This portion is well illustrated with pictures of important and current equipment used by the dispensing and manufacturing pharmacist. The historical portions are particularly well discussed. The specific preparations are discussed in considerable detail. Most of the chapters are well documented, and there is an adequate index. This textbook, in its new edition, should continue to find an important place in the instruction of students of pharmacy and will be a valuable guide to the pharmacist in the services.—*PAUL K. SMITH, Col., USAFR*

RECENT ADVANCES IN NEUROLOGY AND NEUROPSYCHIATRY, by Sir *Russell Brain,* Bt., D. M. (Oxon.), P. R. C. P., and *E. B. Strauss,* M. A., D. M. (Oxon.), D. Sc., F. R. C. P., with the assistance of *Denis Hill,* M. B., F. R. C. P., D. P. M., *Douglas Northfield,* M. S., F. R. C. S., and *David Sutton,* M. D., M. R. C. P., F. F. R., D. M. R. D. 6th edition. 311 pages; 43 illustrations. Little, Brown & Co., Boston, Mass., 1955. Price $7.50.

The current edition of this very stimulating and informative summary of recent advances, principally in the field of neurology and neurophysiology rather than in psychiatry, continues to maintain and improve on the usual high standards employed by these authors in all their writings. The text is a beautiful example of intelligent and "commonsense" summarizing of up-to-date and recent advances in these fields, which considerably reduces the confusion experienced by those who attempt to review the literature on their own. Furthermore, the text has been entirely rewritten with the inclusion of new chapters on electroencephalography and neuroradiology. The authors stress the increasing importance of the relationship of psychiatry to neurology and the need for psychiatrists to possess neurologic knowledge, as continuing progress in neurophysiology, electroencephalography, clinical neurology, and neurosurgery emphasizes the common ground between them.

By admission, the authors lay no claim to comprehensive covering of all subjects and have limited themselves to careful selection of those advances having important clinical bearing or application. This goal has been admirably attained. Unlike the usual yearbooks which merely abstract the literature with all too little discussion and/or continuity, this text is complete within itself, and furnishes sufficient historical background to permit the reader to comprehensively grasp the subjects discussed. It is to be lamented that 10 years have elapsed since the last revision, and it is hoped that widespread appreciation for the yeoman service performed by these authors will be forthcoming and sufficiently rewarding to permit more frequent editions in the future. Certainly the busy practicing neurologist as well as the student will bless them for their efforts. The chapters on the frontal lobes, cervical disk and spondylosis, and electroencephalography seemed particularly lucid and valuable.—*RICHARD R. CAMERON, Col., MC, USA*

THE DISEASES OF OCCUPATIONS, by *Donald Hunter,* M. D., F. R. C. P. 1,046 pages; illustrated. Little, Brown & Co., Boston, Mass., 1955. Price $20.

This new monograph reviews on a broad basis the relationship of disease to occupation, considering nearly every conceivable type of work. In a somewhat disjointed but intriguing way, the opening chapters deal with some of the historical, social, and economic backgrounds of human occupations. The subject has been established on an academic footing by tracing the origin and evolution of trade and health laws and practices, particularly in England.

A brief bibliography follows each chapter. Because the contents have been drawn from worldwide publications, the occupational diseases covered are representative of worldwide experience in this field. The presentation includes historically obsolete health conditions as well as current conditions and those originating from use of new chemicals and processes.

The book is handsomely bound and written in an interesting, easy-to-read style.

The author, in his stimulating treatment of the subject, lays a good foundation for a further study of the etiology, epidemiology, and prevention of occupational disease. He has succeeded admirably in relating environment to disease but does not discuss the patient to the extent often observed in other texts on this subject. Some of the illustrative material seems to be superfluous but most of it is well presented for the student or physician who is being introduced to this field.

Although this book was designed primarily for use by the student, general practitioner, and consultant, the industrial medical officer and industrial hygiene engineer will find it an excellent adjunct to their libraries.

The specialist industrial medical officer and industrial hygiene engineer, on the other hand, will need to consult other books that are more detailed. In some instances, where work hazards are related to such materials as chlorinated hydrocarbons and lead, control measures do not sufficiently emphasize the importance of the periodic physical examination in preventing occupational disease. With such other materials as TNT and nitroglycerine, the importance of minimizing the exposure of the individual person to the toxic agent if adverse effects are to be avoided is not sufficiently stressed.

—*EDWARD J. DEHNE, Lt. Col., MC, USA*

OBSTETRICAL ROENTGENOLOGY, by *Robert Berman,* M. D., F. A. C. S. Second volume of a series of Monographs on Obstetrics & Gynecology, edited by *Claude E. Heaton,* M. D. 599 pages; 486 illustrations. F. A. Davis Co., Philadelphia, Pa., 1955. Price $12.50.

This book is intended to be used by physicians who are actively interested in the subject of obstetrics and should be in the library of

all physicians who are engaged in the practice of obstetrics and roentgenology. In this book the author has covered the entire field of roentgenology as it applies to the products of conception and the bony passageway through which they must pass. This includes the determination of the size and shape of the maternal pelvis; the size, position, number, and development of the fetus or fetuses; and the position within the uterus of the placenta. The author has gone into detail to compare the different methods of x-ray pelvimetry commonly in use today, has pointed out their good and bad features, and has presented his modification of one of these methods. The book is filled with numerous excellent reproductions of roentgenograms. In addition he has covered the technical points and equipment which enter into the production of the roentgenograms and the methods which are used to correct for magnification and distortion. It is felt that the author has done an excellent job of covering this subject, and it is thought that every radiologist and obstetrician will gain in knowledge by reading this book.— *JOHN S. FEATHERSTON, Lt. Comdr., MC, USN*

THERAPY OF FUNGUS DISEASES, An International Symposium, edited by *Thomas H. Sternberg*, M. D., and *Victor D. Newcomer*, M. D. Presented June 23, 24, 25, 1955, by The Division of Dermatology, Department of Medicine, School of Medicine and Medical Extension, University Extension, University of California at Los Angeles. 337 pages; illustrated. Little, Brown and Co., Boston, Mass., 1955. Price $7.50.

This book consists of 54 papers presented by scientists from all over the world at an international symposium held under the auspices of the Division of Dermatology, School of Medicine and Medical Extension, University of California at Los Angeles. This symposium was held to stimulate an exchange of ideas on both a national and an international level concerning the therapy of fungus infections. A total of 208 scientists participated, representing 24 states and 8 foreign countries.

Both the superficial and deep mycotic infections are covered in the papers. Several of the papers report on the status of fungus infections in such countries as India, France, Argentina, Brazil, Mexico, the Philippines, and the Ukraine. The basic biology of fungi, including host-parasite relationship, influence of hormones, soil as their natural reservoir, immunity, nutrition, genetics, et cetera, is discussed.

Many of the papers report on experimental trials in vitro and in vivo, both in animals and humans, of newer drugs such as the aromatic diamidines, cinnamic acids, nitrostyrenes, sulfonamide and sulfone compounds, chlorquinaldol, rhodanine, antihistamines, and the antibiotics filipin and nystatin. There were 16 papers on nystatin alone. The introductory remarks by Dr. Donald M. Pillsbury present many basic problems in the investigative approach to the therapy of fungus diseases.

The attempts at therapy, as presented in the papers themselves, reflect the general concern of the writers for the basic problems of

physiology, biochemistry, and pathology of fungus diseases and, therefore, may well indicate the path of future research.

The book is well illustrated with graphs that are easily understood. It is well worth reading by dermatologists and should be in all reference libraries.—*KARL V. KAESS, Capt., MC, USN*

THE SURGICAL TECHNIC OF ABDOMINAL OPERATIONS, by *Julius L. Spivack,* M. D., LL. D., F. A. C. S. 5th edition. 931 pages; 1,037 illustrations on 495 figures. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $17.50.

This text is essentially a compilation of undergraduate lectures by the author, as presented at the University of Illinois College of Medicine. The subject matter is well organized and presented. The clear and adequate illustrations add tremendously to the elucidation of the procedures. A brief historical review of each section adds greatly to the interest and corrects many popular fallacious beliefs. For each section a brief but adequate review of the essential surgical anatomy is presented. Usually several different procedures to accomplish the same result are described, with the advantages and disadvantages of each enumerated. The chapters on the peritoneum and malformations of the intestinal tract are excellent and timely.

Some surgeons will want to be more radical in handling malignancies, particularly of the left colon, than is recommended by the author. The illustrations for repair of indirect inguinal hernia give the erroneous impression that the hernial sac is outside the spermatic cord. Several inconsequential errors in composition were noted; and at one point several lines are interchanged, which leads to considerable confusion. These errors should have been caught by more careful proofreading.

It is hard to draw a hard and fast line, but if we are to invade the the realm of the gynecologist, one wonders if a work such as this should not include procedures on the kidney, ureter, bladder, adrenals, abdominal sympathetics, and great vessels, which certainly may be attacked by way of the abdomen.

In spite of these possible shortcomings, the clarity of the text and illustrations, plus adequate step-by-step detail, should make this book particularly valuable to the student, intern, and resident learning surgical technic, as well as a valuable reference for the busy surgeon who wishes to refresh his memory before undertaking a surgical procedure.

—*PAUL E. SPANGLER, Capt., MC, USN*

SPLENIN A IN RHEUMATIC FEVER, The Testing of Splenin A as an Anti-inflammatory Agent, by *Alvin F. Coburn,* M. D., *Lucile V. Moore,* M. D., *Judith Wood,* M. D., and *Mary Roberts,* R. N. 87 pages. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $3.75.

This book describes a study of the anti-inflammatory effect of Splenin A which was used in 47 cases of rheumatic fever in children. The method of making this splenin extract is described, a standard unit set up, and

a preparation usable intramuscularly and intravenously developed. Difficulties arose in making a product of lasting stability. The longest follow-up on the patients was three years, in which time they showed no adverse effect from the extract.

The effect on rheumatic fever of some substance in the spleen was postulated when it was noticed that splenectomy had an immediate adverse effect on the course of rheumatic fever in a patient with this disease. Two mutually antagonistic substances, named Splenin A and B, were found in the spleen by Ungar. Splenin A reduces capillary permeability and bleeding time and is found in rheumatic fever patients but not in those with rheumatic arthritis.

Clinical observations in the treated patients indicated an anti-inflammatory effect from Splenin A. However, the difficulties of producing a sufficient amount of the substance, which is not stored in the body in any quantity, and the small numbers of patients in this uncontrolled series leave Splenin A as a very interesting but not presently useful factor. As used in the past, splenic extracts of various sorts, like Pandora's box, have been the source of many disappointments, but hope still remains.—*JAMES L. TOBIN, Col., USAF (MC)*

CHLORPROMAZINE AND MENTAL HEALTH, Proceedings of the Symposium Held Under the Auspices of Smith, Kline & French Laboratories, June 6, 1955, Warwick Hotel, Philadelphia, Pa. 200 pages; illustrated; 25 tables. Lea & Febiger, Philadelphia, Pa., 1955. Price $3.

This handy-sized, well-printed book contains a verbatim report of the papers presented and the discussion offered at a symposium held on chlorpromazine in June 1955 under the auspices of the manufacturer of the drug. Over 100 invited psychiatrists participated in this discussion. The discussants are, for the most part, very enthusiastic about this drug and support their remarks with adequate statistical material. However, there is one criticism one might lay against some of the discussants and that is, that although they usually had significant figures when dealing with schizophrenia, they included in the same tables material from the less common categories of mental disease in which the figures were really too small to be handled statistically.

Many of the projects described seem to have been controlled only by previous experience in the same ward setting. However, there were some other studies that seem to have been carefully controlled by the so-called "double blind" technic. In some of these reports the statistics were not as impressive: in one small group reported by one discussant, placebos, in the opinion of the ward attendants, had been more successful.

However, the general concensus of all the observers was that something very significant and frequently startling happened when chlorpromazine therapy was instituted. The change was particularly striking in the "back ward" situation.

There is an index at the end of the book but it is rather short and probably should be expanded into a book that could be used as a reference. For instance, the reference on "dosage" is divided into only two categories, whereas five or six different listings would be welcomed by the physician picking up the book for help in deciding what dosage should be used for a particular case.

This book is recommended for all those who wish to refer to the most recent material published in book form on this most important subject of the chemical approach to mental disease, a subject which is having such a tremendous vogue at the present time.

—IRA C. NICHOLS, Capt., MC, USN

PSYCHOSOMATICS, by *Max Hamilton,* M. D., D. P. M. With a Foreword by Professor *D. R. MacCalman,* M. D., F. R. C. P. E. 225 pages; illustrated. John Wiley & Sons, Inc., New York, N. Y., 1955. Price $4.25.

This book is a critical review of the literature on psychosomatics of the past 20 years. The author has selected writings largely by British and American authors, though some of the more important work in languages other than English is reviewed too. Much of the material has been selected for its suitability for testing by statistical methods. Although there is a chapter each on physiology of the emotions, the respiratory system, the cardiovascular system, the gastro-intestinal system, skin disorders, endocrines and metabolism, and general problems and conclusions; disappointingly, there is no section on neuromuscular disorders or genito-urinary disorders, and relatively little discussion of the psychologic aspects of certain diseases, such as coronary disease, which admittedly have an organic basis but in which psychologic factors may play an important part.

Nevertheless, this book contains a wealth of factual information on psychosomatics, and is written in a clear and fluent style. It contains much information of interest not only to the psychiatrist but also to the internist, allergist, and medical student. For those who need a short reference work on this subject, this book fills that need admirably.

Of special interest is the last chapter, in which the author discusses the strengths and weaknesses of certain theoretic considerations of psychosomatic illness. One of the striking features of the personality studies undertaken in psychosomatic illnesses is the rather remarkable similarity of the personality structures of persons suffering from rather widely dissimilar physical manifestations. This should give pause to those dynamic psychiatrists whose orientation tends to disregard somatic manifestations, and should emphasize further that research in this field must go beyond mere tabulation of personality traits in relation to various diseases. In conclusion, this little book fills a definite need for periodic, systematic reviews of recent literature, and in the opinion of the reviewer is well worth the purchase price.

—ROY E. CLAUSEN, Jr., Lt. Col., MC, USA

New Books Received

Books received by the *U. S. Armed Forces Medical Journal* are acknowledged in this department. Those of greatest interest will be selected for review in a later issue.

COLLECTED PAPERS ON AVIATION MEDICINE. AGARDograph No. 6. Presented at Aeromedical Panel Meetings of the Advisory Group for Aeronautical Research and Development, Palais de Chaillot, Paris. Published for and on behalf of Advisory Group for Aeronautical Research and Development, North Atlantic Treaty Organization. 218 pages. Interscience Publishers, Inc., New York, N. Y., 1955. Price $5.

A Clinical, Pathological, and Genetic Study of MULTIPLE NEUROFIBROMATOSIS, by *Frank W. Crowe,* M. D., *William J. Schull,* Ph. D., and *James V. Neel,* M. D., Ph. D. This Study was supported by Contract AT (11-1)-70 from The United States Atomic Energy Commission. American Lecture Series, Publication No. 281, A Monograph in The Bannerstone Division of American Lectures in Dermatology, edited by *Arthur C. Curtis,* M. D. 181 pages; illustrated. Charles C Thomas, Publisher. Chicago, Ill., 1956. Price $5.

MODERN OPERATIVE SURGERY, in Two Volumes, Volume I, edited by the late *G. Grey Turner,* LL. D., D. Ch., M. S., F. R. C. S., F. R. A. C. S., F. A. C. S., and *Lambert Charles Rogers,* V. R. D., M. Sc., M. S., F. R. C. S., F. R. C. S. E., F. R. A. C. S., F. A. C. S., with a foreword by Sir *Gordon Gordon-Taylor,* K. B. E., C. B., M. A., LL. D., Sc. D., M. D., M. S., F. R. C. S., F. R. C. S. E., F. R. C. S. I., F. R. A. C. S., F. A. C. S. 4th edition. 1,229 pages; illustrated. Paul B. Hoeber, Inc., New York, N. Y., 1956. Price $15.

The Diagnosis and Treatment of POSTURAL DEFECTS, by *Winthrop Morgan Phelps,* M. D., *Robert J. H. Kiphuth,* M. P. E., and *Charles Weer Goff,* M. D. 2d edition. 190 pages; illustrated. Charles C Thomas, Publisher, Springield, Ill., 1956. Price $6.50.

HYPOTHALAMIC-HYPOPHYSIAL INTERRELATIONSHIPS, A Symposium, Third Annual Scientific Meeting of the Houston Neurological Society, Texas Medical Center, Houston, Tex. Compiled and edited by *William S. Fields, Roger Guillemin,* and *Charles A. Carton.* 156 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $4.75.

PROGRESS IN CLINICAL PSYCHOLOGY, Volume II, edited by *Daniel Brower,* Ph. D., and *Lawrence E. Abt,* Ph. D. 364 pages. Grune & Stratton, Inc., New York, N. Y., 1956. Price $7.75.

NORADRENALINE, Chemistry, Physiology, Pharmacology and Clinical Aspects, by *U. S. von Euler,* M. D., F. A. C. P. (Hon.). American Lecture Series, Publication No. 261, A Monograph in The Bannerstone Division of American Lectures in Physiology, edited by *Robert F. Pitts,* M. D., Ph. D. 382 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $11.50.

THE TRUTH ABOUT CANCER, by *Charles S. Cameron,* M. D. 268 pages. Prentice-Hall, Inc., New York, N. Y., 1956. Price $4.95.

SUBPHRENIC ABSCESS, by *H. R. S. Harley,* M. S., F. R. C. S. American Lecture Series, Publication Number 255, A Monograph in The Bannerstone Division of American Lectures in Surgery, edited by *Michael E. DeBakey,* M. D., and *R. Glen Spurling,* M. D. Thoracic Surgery Division, edited by *Brian Blades,* M. D. 216 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1955. Price $7.

SYNOPSIS OF GYNECOLOGY, Based on the Textbook "Diseases of Women," by *Robert James Crossen,* M. D., F. A. C. S. 4th edition. 255 pages; 132 illustrations, including frontispiece in color. The C. V. Mosby Co., St. Louis, Mo., 1956. Price $5.25.

THE CERVICAL SYNDROME, by *Ruth Jackson,* M. D., F. A. C. S. American Lecture Series, Publication No. 268, A Monograph in American Lectures in Orthropedic Surgery, edited by *Robert J. Joplin,* M. D. 130 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $4.75.

The Laboratory Diagnosis of COAGULATION DEFECTS, by *Pietro De Nicola,* M. D. American Lecture Series, Publication No. 269, A Monograph in The Bannerstone Division of American Lectures in Pharmacology, edited by *Chauncey D. Leake,* Ph. D. 240 pages; 62 illustrations and 26 tables. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $7.50.

STRABISMUS, Diagnosis and Treatment, by *Beulah Cushman,* M. S., M. D. 208 pages; illustrated. Lea & Febiger, Philadelphia, Pa., 1956. Price $6.

SKIN SURGERY, by *Ervin Epstein,* M. D., and 17 contributors. 228 pages; 242 illustrations on 101 figures. Lea & Febiger, Philadelphia, Pa., 1956. Price $7.50.

Clinical Recognition and Management of DISTURBANCES OF BODY FLUIDS, by *John H. Bland,* M. D. 2d edition. 522 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1956.

ELECTROCARDIOGRAPHY, Fundamentals and Clinical Application, by *Louis Wolff,* M. D. 2d edition. 342 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1956. Price $7.

THE NEUROSES IN CLINICAL PRACTICE, by *Henry P. Laughlin,* M. D. 802 pages. W. B. Saunders Co., Philadelphia, Pa., 1956.

SURVEY OF CLINICAL PRACTICE IN PSYCHOLOGY, edited by *Eli A. Rubinstein,* Ph. D., and *Maurice Lorr,* Ph. D. Foreword by *Laurance F. Shaffer.* 363 pages. International Universities Press, Inc., New York, N. Y., 1954. Price $6.

DOCTOR AND PATIENT AND THE LAW, by *Louis J. Regan,* M. D., LL. B. 3d edition. 716 pages. The C. V. Mosby Co., St. Louis, Mo., 1956. Price $12.50.

THE ANNUAL SURVEY OF PSYCHOANALYSIS, A Comprehensive Survey of Current Psychoanalytic Theory and Practice. Volume III. Edited by *John Frosch,* M. D., *Nathaniel Ross,* M. D., *Sidney Tarachow,* M. D., and *Jacob A. Arlow,* M. D. 682 pages. International Universities Press, Inc., New York, N. Y., 1956. Price $10.

"CONTROLLED HYPOTENSION" in ANESTHESIA and SURGERY, by *David M. Little, Jr.,* M. D. American Lecture Series, Publication No. 283, A Monograph in The Bannerstone Division of American Lectures in Anesthesiology, edited by *John Adriani,* M. D. 159 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $4.50.

POLICE DRUGS, by *Jean Rolin*, translated, with a foreword, by *Laurence J. Bendit*, M. A., M. D. (Cantab.), D. P. M. With an appendix on Narcoanalysis by *Edward V. Saher*. 194 pages. Philsophical Library, New York, N. Y., 1956. Price $4.75.

LABORATORY TESTS IN COMMON USE, by *Solomon Garb*, M. D. 160 pages. Springer Publishing Co., Inc., New York, N. Y., 1956. Price $2.

CLINICAL LABORATORY METHODS AND DIAGNOSIS, Volumes One and Two, by *R. B. H. Gradwohl*, M. D., D. Sc. 5th edition. Volume I, Chapters I to VI, pages 1-1220, 29 color plates, 295 figures. Volume II, Chapters VII to XVII, pages 1221-2452, 23 color plates, 470 figures. The C. V. Mosby Co., St. Louis, Mo., 1956. Price $38.50 for two volumes.

THE YEAR BOOK OF UROLOGY (1955-1956 Year Book Series), edited by *William Wallace Scott*, M. D., Ph. D. 398 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1956. Price $6.50.

DISEASES OF THE NERVOUS SYSTEM, by Sir *Russell Brain*, Bt., D. M. (Oxon.), P. R. C. P. (London). 5th edition. 996 pages; illustrated. Oxford University Press, New York, N. Y., 1955. Price $10.50.

METABOLISM, PHARMACOLOGY AND THERAPEUTIC USES OF GOLD COMPOUNDS, by *Walter D. Block*, Ph. D., and *Kornelius Van Goor*, M. D. American Lecture Series, Publication No. 282, A Monograph in American Lectures in Dermatology, edited by *Arthur C. Curtis*, M. D. 76 pages. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $2.75.

CLINICAL STUDIES IN NEUROLOGY, by *Harry Lee Parker*, M. S., M. D., F. R. C. P. I. 362 pages. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $6.50.

PROGRESS IN NEUROBIOLOGY: I. NEUROCHEMISTRY, edited by *Saul R. Korey*, M. D., and *John I. Nurnberger*, M. D., with twenty-three contributors. 244 pages; illustrated. Paul B. Hoeber, Inc., Medical Book Dept. of Harper & Bros., New York, N. Y., 1956. Price $6.75.

GESTATION, Transactions of the Second Conference, March 8, 9 and 10, 1955, Princeton, N. J., edited by *Claude A. Villee*, Ph. D. 262 pages; 118 illustrations. Josiah Macy, Jr. Foundation, New York, N. Y., 1956. Price $5.

THE NONVENEREAL DISEASES OF THE GENITALS, Etiology, Differential Diagnosis and Therapy, by *Fritz T. Callomon*, M. D., and *John F. Wilson*, M. D., M. S. 382 pages; 150 illustrations. Charles C Thomas, Publisher, Springfield, Ill., 1956. Price $12.50.

AMERICAN MEN OF SCIENCE, A Biographical Directory, edited by *Jaques Cattell*. 9th edition. Volume II, "Biological Sciences." 1,276 pages. The Science Press, Lancaster, Pa., and R. R. Bowker Co., New York, N. Y., 1955. Price $20.

THE NAVAL OFFICER'S MANUAL, by Rear Admiral *Harley Cope*, USN (Retired). 3d edition. A Ready Reference of Helpful Information and Counsel for All Officers of the United States Navy and the Marine Corps. 608 pages; illustrated. The Military Service Publishing Co., Harrisburg, Pa., May 1955. Price $4.

BRITISH MEDICAL BULLETIN, Volume 11, Number 3, September 1955, "Surgery of the Heart and Thoracic Blood Vessels." 242 pages; illustrated. Published by the Medical Dept., The British Council, London W1, England. Distributed by Oxford University Press, New York, N. Y., 1955. Price $2.75.

BRITISH MEDICAL BULLETIN. Volume 12, Number 1, January 1956, "Recent Research on Vitamins." 90 pages; illustrated. Published by the Medical Dept., The British Council, London W1, England. Distributed by Oxford University Press, New York, N. Y., January 1956. Price $2.75.

PRINCIPLES OF GENERAL PSYCHOLOGY, by *Gregory A. Kimble.* 400 pages; 211 illustrations; 8 tables. The Ronald Press Co., New York, N. Y., 1956. Price $5.

A TEXTBOOK OF ENDODONTOLOGY, by *Edgar D. Coolidge,* M. S., D. D. S., LL. D. (Hon. Loyola), and *Robert G. Kesel,* D. D. S., M. S. 2d edition. 366 pages; 345 illustrations on 210 figures and 1 plate in color. Lea & Febiger, Philadelphia, Pa., 1956. Price $7.50.

UROLOGICAL SURGERY, by *Austin Ingram Dodson,* M. D., F. A. C. S., with nine contributors. 3d edition. 868 pages; 664 illustrations. The C. V. Mosby Co., St. Louis, Mo., 1956. Price $20.

SURVEY OF CLINICAL PRACTICE IN PSYCHOLOGY, edited by *Eli A. Rubinstein,* Ph. D., and *Maurice Lorr,* Ph. D. Foreword by *Laurance F. Shaffer.* 363 pages. International Universities Press, Inc., New York, N. Y., 1954. Price $6.

COLOR ATLAS OF ORAL PATHOLOGY, Histology and Embryology, Developmental Disturbances, Diseases of the Teeth and Supporting Structures, Diseases of the Oral Mucosa and Jaws, Neoplasms. Prepared under the auspices of the *U. S. Naval Dental School,* National Naval Medical Center, Bethesda, Maryland. 188 pages; 461 figures in color. J. B. Lippincott Co., Philadelphia, Pa., 1956. Price $12.

IN THE DOCTOR'S OFFICE, The Art of the Medical Assistant, by *Esther Jane Parsons.* Illustrations by *Jean McConnell.* 2d edition. 326 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., 1956. Price $3.95.

ESSENTIALS OF DERMATOLOGY, by *Norman Tobias,* M. D. 5th edition. 651 pages; 211 figures; 11 subjects in color. J. B. Lippincott Co., Philadelphia, Pa., 1956. Price $8.

THORACIC SURGERY FOR PHYSIOTHERAPISTS, by *Gladys M. Storey,* S. R. N., with a foreword by *N. R. Barrett,* M. Chir. 132 pages; illustrated. J. B. Lippincott Co., Philadelphia, Pa., 1955. Price $3.

ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, Volume 63, Article 5, pages 637-1030. Editor: *Roy Waldo Miner.* "Ascites Tumors as Tools in Quantitative Oncology," by *T. S. Hauschka* and 40 investigators. 393 pages; illustrated. The New York Academy of Sciences, New York, N. Y., March 14, 1956. Price $4.50.

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